VOLUME XXI AUGUST 1928 NUMBER 8

Southern Medical Journal

Journal of the Southern Medical Association

Published monthly by the Southern Medical Association, Empire Building, Birmingham, Ala. Annual subscription $4.00. Entered as Second-Class Matter at the Post Office at Birmingham, Ala., under Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of Oct. 3, 1917, authorized Dec. 20, 1921.

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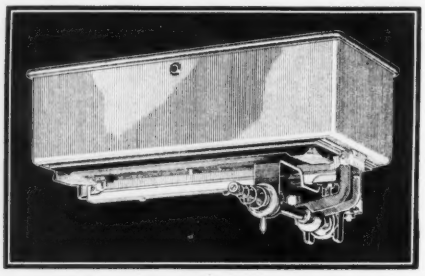

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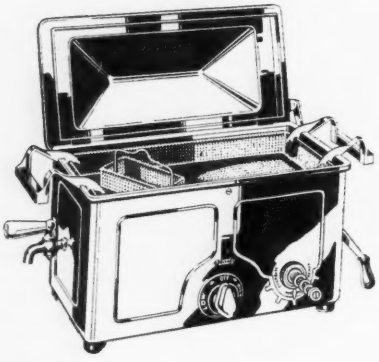

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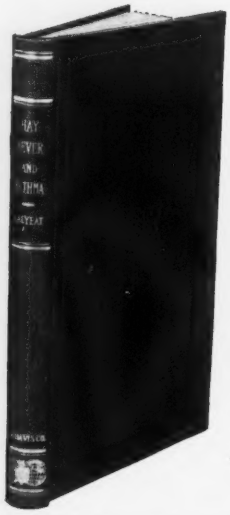

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"To this end the committee has collaborated with a large number of laboratories and clinics in the United States and abroad which have been interested in one or another of the clinical, chemical or biological problems that have arisen.

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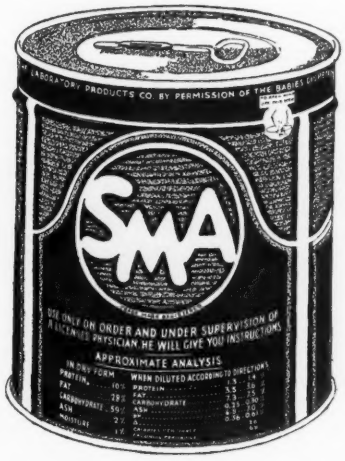

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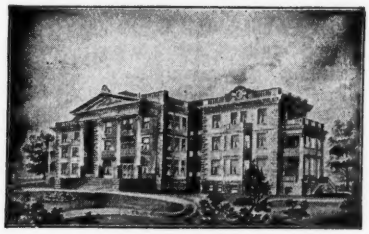

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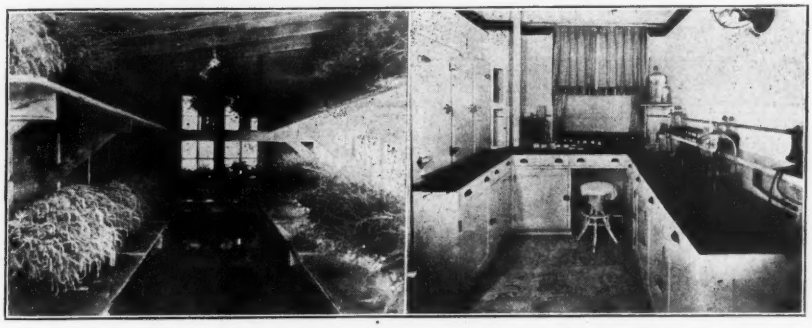

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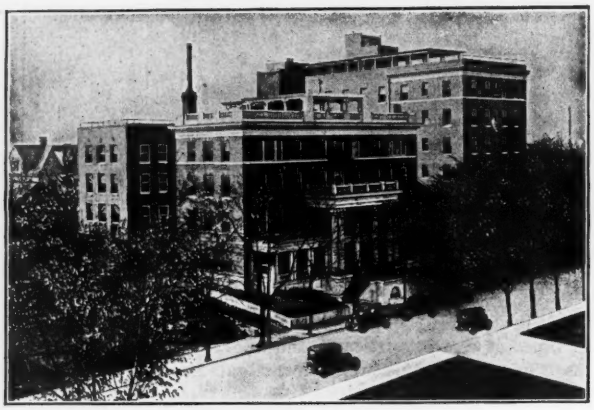

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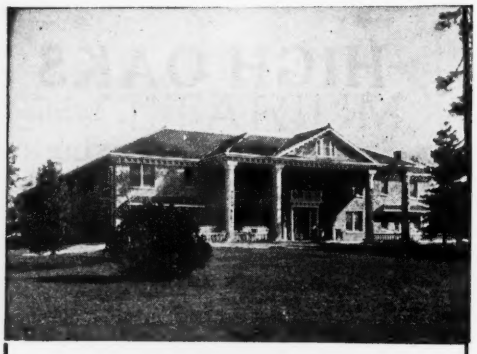

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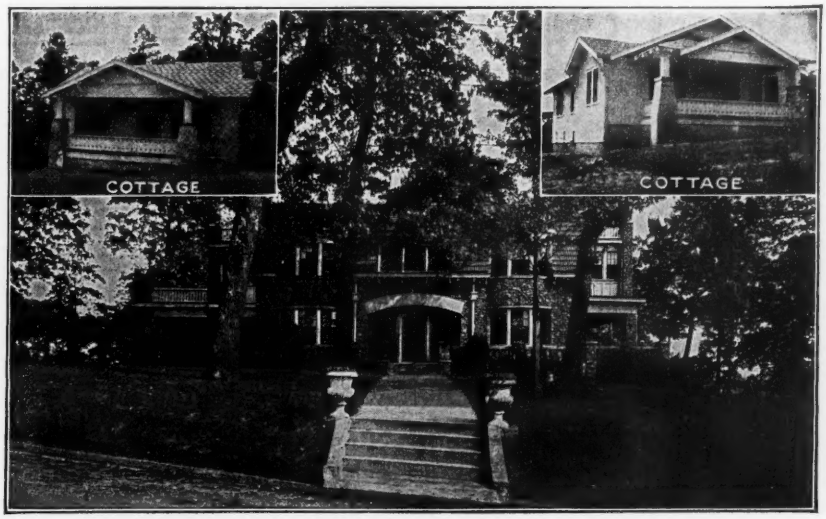

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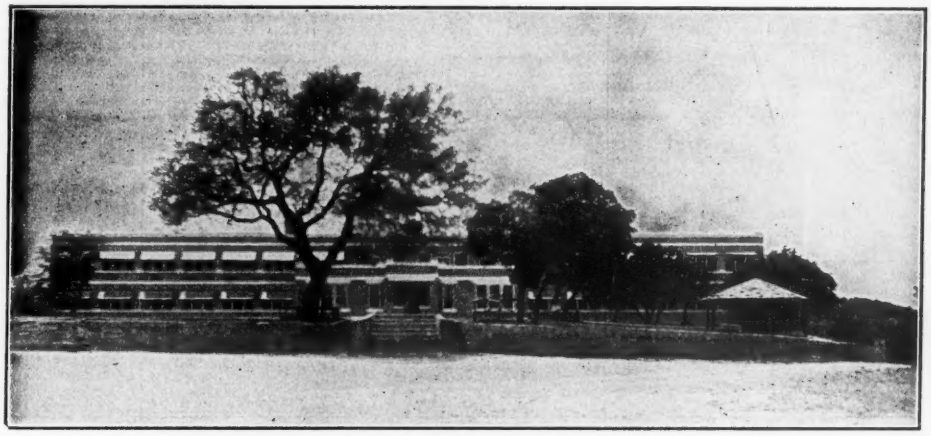

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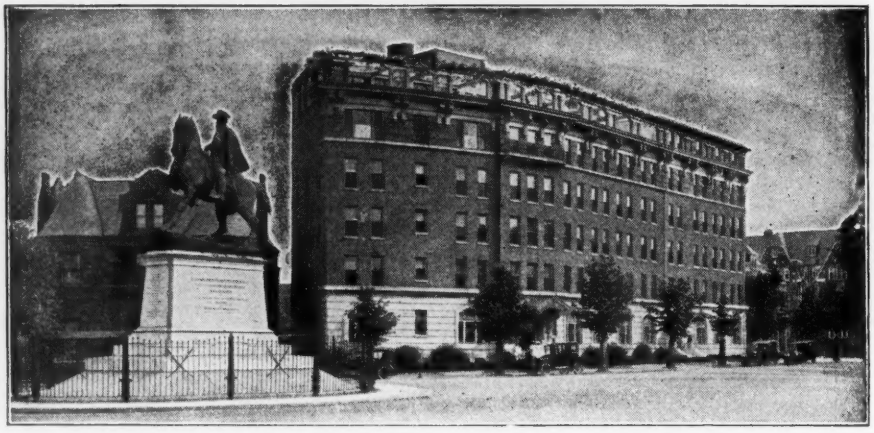

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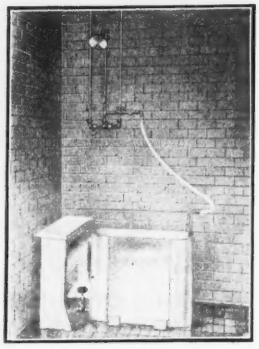

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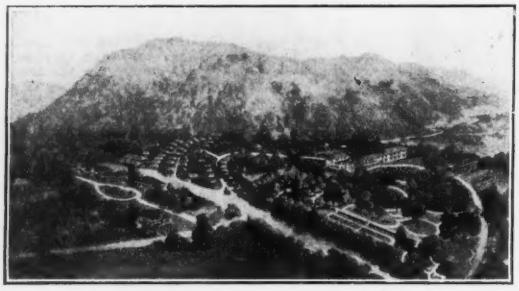

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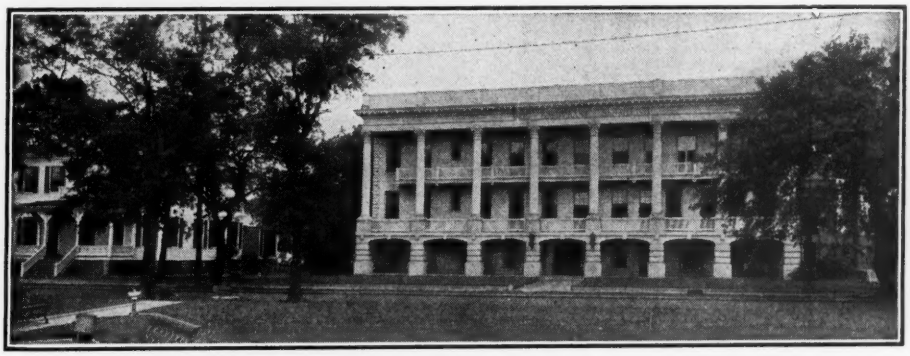

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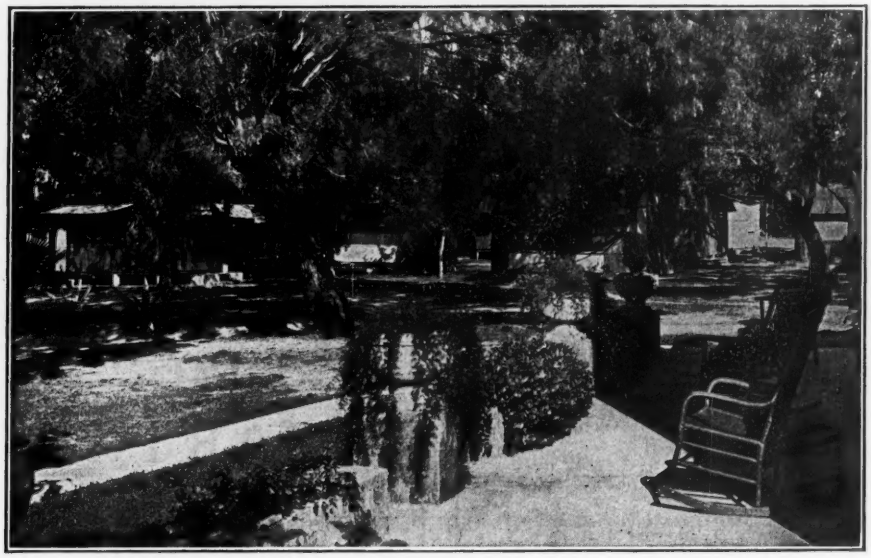

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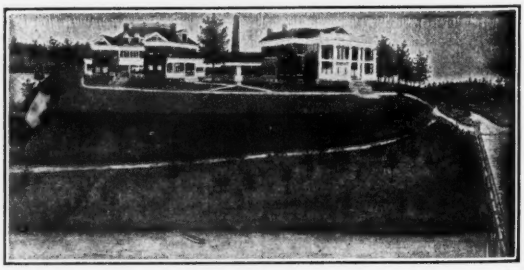

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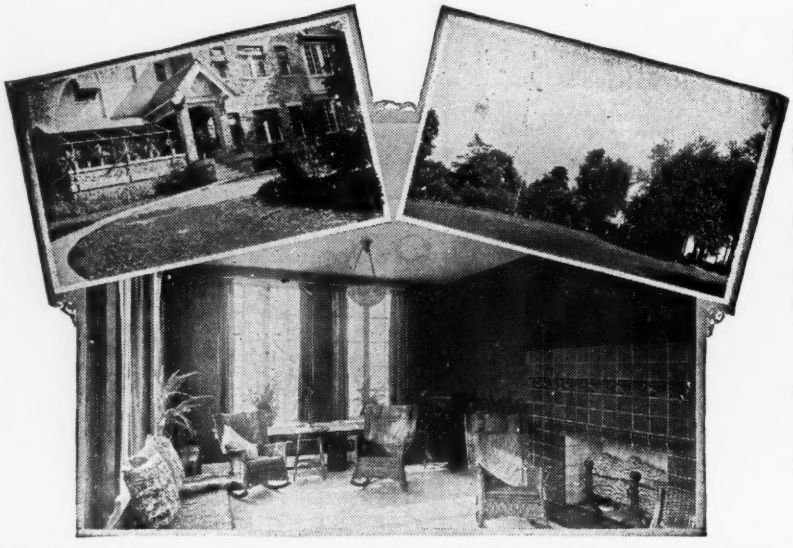

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Volume XXI AUGUST 1928 Number 8

HISTOLOGICAL CHANGES IN THE MAMMARY GLANDS OF ANIMALS FROM INVERSION OF THE NIPPLE*†‡

By WILLIAM H. HARRIS, M.D.,
New Orleans, La.

Inverted and inflammed nipples of the mammary gland of the female and chronic mastitis have long been considered as common precursors of carcinoma of this structure. It is believed that these conditions supply the tissue changes regarded by Ribbert as a causative factor of carcinoma, namely, disturbance of proper epithelial cell relationship and abnormal proliferation of such cells. Such histological disturbances occasion the segregation of epithelium from its normal physiological control, permit aberrant and wild growth and thus produce the histological, parasitic neoplasm known as carcinoma. With such features as a basis it was thought that the duplication of these conditions experimentally would lead to the production of carcinomatous growth.

Experiments were first carried out upon female rabbits.[1] In these animals a three-sided flap was made with the nipple surmounting this detached portion. The nipple proper was scarified and pruned. A bed was prepared in the underlying structure in which was embedded the nipple and its usual small amount of gland structure. This was accomplished by the passage of a suture through the skin above the flap, next through the flap and thence returning near the point of entrance. By pulling upon this loop and tying the ends, the nipple flap was maintained in this abnormal situation. The skin over the gaping area from which the flap was inverted was then sutured together. This procedure with slight variations was carried out upon eight rabbits for two or more of the nipples. Twenty-eight such operations were performed. Numerous nodular masses in part inflammatory, in part proliferative, were formed by such procedures. Some of these increased in size and persisted weeks or months, others eventually receded and underwent fibrosis.

Portions of such nodules were removed from time to time for histological study. The changes revealed many features of interest. The epithelium both of the inverted skin and hair follicles as well as that of gland proper displayed marked evidences of proliferation. In the skin, the epithelium showed a tendency to "pearl" formations and many islands of cells were segregated from their original location, lying deep in the corium. The hair shafts continued to grow, quite often in a downward or lateral direction. The sebaceous glands continued to secrete and as this secretion was not discharged a dermoid cyst-like formation often resulted. The glandular epithelium formed at times papillary protrusions within the ducts and papillo-cystomatous masses resulted. In other nodules the glands became hyperplastic at times with rupture of the basement membrane, giving a carcinoma-like histological appearance. Although they were observed for months, no metatasis was ever found and the growths either remained dormant, formed cyst-like masses or underwent absorption or fibrosis.

As cancer of the breast of the rabbit is an unusual occurrence, it was thought that the inherent natural resistance of this species of animal to such neoplasms might be the reason for the failure of cancer production. It was considered that some other type of animal in which cancer of the breast occurs spontaneously, might form a more suitable field for such experimentation. As female dogs were known to be victims of cancerous neoplasms of the breast,[2] dogs were selected for further experimentation. Seven female dogs of unknown age, apparently four or more years old, were em-

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*Read in Section on Pathology, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

†From the Department of Pathology of the Tulane University, School of Medicine.

‡Aided by a grant from the B. M. Harrod Cancer Research Fund.

ployed. Two or more nipples were selected, using approximately thirty such areas. The breasts were well developed and little if any hair present over the nipple area. The procedure was somewhat different from that in the rabbit because of the structure with which to work was better. An ellipitical incision was made about the nipple and the skin covering the area within the incision, including that of the nipple, was removed. The nipple stump was pruned and scarified. A bed was prepared down through the breast tissue and into the deep underlying structures. In this manner the breast was somewhat macerated. A deep suture was introduced through the skin at the base of the gland and then passed up from the depth of the "bed" through the nipple flap and returned along this same path to near the point of entrance. Traction on the suture drew the nipple and parts of the breast well down into the deeper area; the suture was tied thus retaining the flap in position. The overlying edges of the wound were sutured together. Through this means nodules of varying sizes were produced. Many of these increased in size for weeks and months. Inflammatory changes were more extensive than in the rabbit nodules and in many instances considerable suppuration occurred. Portions of these nodules were removed at varying periods for microscopic study. The results obtained through these measures were somewhat analogous to those obtained for the rabbit except that changes were more marked and the proliferations more extensive, probably because of the more favorable character of tissue present in the dog breast. No epidermal or hair growths were present in these instances as the skin and its appendages had been removed from the flap.

SUMMARY AND DISCUSSION

In the rabbit and dog experiments here reported, fifteen animals were employed and approximately sixty operations carried out. In the nodules thus produced various types of gland, skin and hair proliferations were noted. Papillary, cystic areas and cell masses were formed. Three of the animals were lactating and in these the adenomatous, cystic and papillary formations were more marked although the general results were similar. In the cancer-like masses formed, mitotic figures were often seen, although the cells as a whole maintained the regular tinctorial and morphological characters present in the previous normal histological structure. Some of the dogs with nodule development, are still under observation and although more than a year has elapsed no true evidences of malignancy have been found.

In this connection, the carcinomas obtained by tar applications are of interest. It would seem that tar has some selective capacity of stimulating the cells to malignant growth. It is not necessarily a local stimulation, as Murphy[3] has shown in certain experiments that tar application to the skin can produce cancer of the lung with no local epidermoid carcinoma. Sharlach R may also produce epithelioma-like changes. Adair and Baggs[4] have produced carcinoma of the breast by disturbing lactation in mice, causing stagnation by various processes. Burrows[5] considers that abnormal crowding of cells and disturbed circulation may affect the vitamins of the tissues and bring about cancer formation. While our reported experiments may not have attained the disturbed balance deemed necessary by Burrows to establish neoplastic epithelial growth, certainly very marked crowding of cells and altered blood circulation were produced.

We know that areas histologically resembling carcinoma may at times be found, as was recently pointed out by White and Weidman,[6] when no true carcinoma exists. In our experiments, areas demonstrating carcinoma from the histologic viewpoint were observed but these did not possess the functional capacity of metastasis found in true carcinoma. It is apparent that there is some intrinsic or inherent capacity newly developed or modified in true carcinoma cells through their own unbalanced or disturbed function. The mechanism of this factor may have been initiated within the cell itself or have been introduced from an extra-cellular source. Once, however, the cell assumes this abnormal or "parasitic character" it perpetuates cells having similar characteristics either at the local site or when they occur as metastatic foci. It is, as it were, a selective abnormal physiological capacity developed by such cells whereby normal function and regulation are lost and the aberrant cells become rebellious invaders of the parent host usually leading to destruction of the organism.

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GIARDIASIS IN CHILDREN*

By JOHN ZAHORSKY, M.D.,
St. Louis, Mo.

It is our custom to examine all diarrheal stools, diluted with water, under the low power of the microscope, to determine the cellular content. On this, in a great measure, the differentiation between fermentative and infectious diarrhea is made. During the summer of 1925 I encountered two cases showing a large number of oval shaped shining bodies, the nature of which was unknown to me. During the summer of 1926 having found the same refractile bodies in the stools of twins, I asked Dr. Mary McLoon to study them, stain them, and ascertain their nature. After considerable difficulty she at last found a typical vegetative form of giardiae, and the stained refractile bodies gave the markings of giardial cysts. It was easy, thereafter, to identify the cysts under the low power of the microscope.

In studing the literature we found that much had been written on the subject of intestinal flagellates, and many protozoolgists had contributed studies on the life history and classification of these organisms. I need only refer to the excellent text book by Craig, "The Parasitic Protozoa in Man."

Special interest of pediatricians in this subject was aroused by two studies of Reginald Miller,[1 2] of London, who concluded that the infection is by no means rare, that it produces a chronic enteritis, and that the resultant diarrhea is often severe enough to retard growth and development.

This flagellate was described by Lambl more than half a century ago, and for a long time was considered a harmless intestinal parasite, but the studies of Miller and others seem to justify the conclusion that it is not a harmless infection to infants. My own experience supports this contention. The presence of this parasite in the intestinal canal of infants produces a chronic irritation of the upper intestinal tract, which results in increased peristalsis, deficient absorption of food and an augmentation of bacterial activity. The resultant fermentation favors diarrheal and gaseous distension of the abdomen and a consequent retardation in growth. Recently, Boeck has combated the theory that giardiae have pathogenic effects, but his observations were confined to older children and adults. In support of my contention I ask you to notice the weights of the infants suffering from giardiasis, which we have studied during the past two years.

Case	Age	Weight	Case	Age	Weight
1	8 mo.	12.5 lbs.	5	32 mo.	27 lbs.
2	8 mo.	12.3 lbs.	6	13 mo.	17 lbs.
3	8 mo.	16. lbs.	7	30 mo.	29 lbs.
4	10 mo.	18. lbs.	8	4 mo.	10.7 lbs.

The greatest effect on weight is found in the young infant. Three older children (*Case 9 and 10*) who were found infected with giardiae had no marked retardation in growth. All these infants had a distinct tendency to meteorism. The abdomen was invariably larger than normal. In four cases this enlargement was so marked as to suggest the presence of celiac disease. In all cases the symptoms indicated abdominal distress. Three older children complained of recurrent abdominal pains. One case was mistaken for chronic appendicitis.

The deficiency in growth is also shown by the flabby musculature, the sallow color, and a tendency to anemia, which was a striking symptom in one of our cases.

I must, therefore, conclude that in the young infant an intestinal infestation by giardiae produces symptoms; and therapeutic measures for the eradication of this infection are clinically indicated.

Symptoms.—The striking symptom in all cases is a tendency to looseness of the bowels. This may vary from day to day, but unless some secondary infection of the intestinal mucosa occurs, no marked diarrhea is present. The stools are thin, soft, or mushy and, contrary to the true enterites, do not contain cells. Our studies, except in three cases in which a secondary infection had taken place, revealed no inflammatory change of the mucosa. There is merely a heightened peristalsis and increased fermentation of the intestinal contents. The stools contain an increased amount of fat and undigested particles.

A prominence of the abdomen is nearly always present, but varies according to the duration of the infection. As already mentioned abdominal pain, attacks of colic, and loss of appetite are present at irregular intervals.

The nutritional state suffers more or less, and the babies have a poor color, the tissues are flabby and the weight is below normal. No marked change has been found in the blood. There is no eosinophilia.

*Read in Section on Pediatrics, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

Diagnosis.—The diagnosis depends on the demonstration of large numbers of giardial cysts in the stool. These cysts are shining refractile bodies, somewhat oval in shape and about the size of a polymorphonuclear leucocyte. The cysts appear structureless, but special staining methods bring out the characteristic markings. We used Kofoid's modification of Donaldson's iodin-eosin stain which brings out the structure of the cysts. The nuclei appear as two eyes in one end of the oval body and are diagnostic. Occasionally one is so fortunate as to find a trophozoite, or vegetative form, of the flagellate which looks like a tadpole and cannot be mistaken.

These cases are usually diagnosed chronic intestinal indigestion or celiac disease and the differentiation depends on the discovery of the intestinal parasite.

Dr. Mary McLoon and I[3] reported three cases before the St. Louis Medical Society one year ago. The twins infected with giardiae, as reported in that paper, have been under observation for more than a year. While the treatment improved the condition, relapses occurred several times. The bismuth treatment had to be instituted again and again. Now, fourteen months after the beginning of the treatment they seem very well, but are still somewhat under the average, in size. When last seen they were seventeen months old and weighed 20½ and 22 lbs. respectively. The prominence of the abdomen was not noticeable in either infant and no giardiae were found in the stools.

REPORT OF CASES

Case 1.—B. W., a boy, 2 years old was seen first Dec. 31, 1926. He was breast fed for several months and seemed to do well until he was about eighteen months old when he had a diarrhea during the summer. This subsided but a tendency to looseness of the bowels persisted. He did not gain in weight. For the preceding two weeks, he had had vomiting attacks immediately after eating.

He had had repeated colds and measles when he was one year old. The baby had a good color, but the tissues were flabby. Examination showed nothing abnormal but a markedly distended abdomen. Hemoglobin was 90 per cent. The stool was very light in color and contained much fat. No cells or cysts in the stool were observed at this time. The weight was 24 pounds.

He was placed on a diet of solid food with no milk. He improved very much during the next three months, although he had one attack of bronchitis. The weight March 24, was 27 pounds. May 19, 1927, the baby had three or four stools a day but no vomiting. The appetite was poor but he continued to gain. The weight was 27½ lbs.

The stool obtained with a catheter was thin but no microscopic examination was made. On June 5, he still had 3 or 4 stools daily which under the microscope showed many cysts of giardiae. He was given bismuth salicylate, 4 grains three times a day. The diet was not changed.

In spite of this treatment he did not gain. He had another attack of the grippe two weeks later. The bismuth made the stools firm and hard but still the cysts were always present in large numbers (10 to 30 to each field of the diluted stool). July 3, 1927, the failure of the bismuth to diminish the giardiae, induced me to try oil of chenopodium, three drops three times a day. It was difficult to give him this and no apparent effect was obtained on the parasite.

July 12, he was again placed on bismuth treatment with almost a complete disappearance of the cysts, but they were again very numerous in the stool a few weeks later.

August 19, he still had two to three stools daily, but the passages were hard. It was found that these hard stools were packed with cysts. He was then given, one grain of stovarsol twice a day. This was increased to four grains daily. The cysts disappeared and he gained a pound in weight. His weight was 29½ lbs.

Case 2.—T. B., a boy 6 years old, was seen August 7, 1927. He had had abdominal pain for several months. The bowels were rather loose, general condition good, stool thin, and giardial cysts very numerous. Five grains of bismuth salicylate were given three times a day. The cysts were not found on two subsequent examinations after three weeks treatment.

Case 3.—M. S., a girl 19 months old was seen Aug. 1, 1927. She had just passed through an attack of whooping cough.

The stools had been a little loose. There were two or three stools a day. The abdomen was somewhat distended. Giardial cysts were numerous. Bismuth salicylate, 3 grains three times a day was given. The cysts disappeared from the stool in ten days, but some were again present three weeks later.

Case 4.—W. M., 4 months old, a boy, was seen Oct. 9, 1927. He was constipated, had a very large abdomen, was poorly nourished, and weighed 11 pounds. The stool was thin, watery, full of giardial cysts, and also trophozoites. Bismuth salicylate was given. He was seen a few months later and no cysts were found.

Case 5.—J. D., a girl 10 months old, was seen Sept. 15, 1927. She had had diarrhea for several days. The stool was full of mucus. She had fever 101°, no vomiting, but typical colitis. The stool under the microscope was full of leucocytes and macrophages. There were many giardial cysts among the cells. This was evidently a case of infectious enteritis complicated by giardiasis. Bismuth salicylate was given.

Case 6.—A girl 14 months old had a tendency to looseness of the bowels, with 2 to 4 thin stools daily. She was small for her age, her weight was 14 lbs. The stools contained numerous giardial cysts, 5 to 10 in each field. Bismuth salicylate was given over a period of two weeks, followed by a disappearance of the cysts and improvement in her general condition. Two months later she had an infectious enteritis but no giardia were found in the stool.

Source and Transmission.—It is generally accepted at present, that human infection with giardiae has its source in another human being. The giardia of animals, according to Simon, are

a distinct species. It is reasonable, therefore, to regard fluids and food contaminated by human feces as the usual means of transmission. As the cysts withstand ingestion by flies, these insects must be considered as possible conveyors of the infection. It is easily seen how the soiled fingers of an attendant may infect the baby. The cysts are somewhat resistant to heat. An exposure for five minutes to 64° C. is required to kill them. It is obvious that the ordinary method of pasteurizing milk either by the "held" method or the "flash" method may not succeed in preventing infection. Cream, so often sold contaminated by flies, may be a source of giardiasis as well as dysentery.

Incidence.—The infection seems to be fairly frequent. Boeck and Stiles in a survey, in which over 8000 persons were examined, reported an incidence of *Giardia intestinalis* of 6.5 per cent.

In England the incidence is much higher judging from a report by Dobell. The incidence in children seems to be higher than in the adult. In Leeds, an examination of the stools of 128 children revealed the presence of giardial cysts in more than one-third of them. We have examined the stools of approximately 500 children showing diarrheal tendencies and found ten cases infected with *Giardia intestinalis.*

Prognosis.—The cysts are killed with great difficulty, and it is not always possible to foretell how long the child will remain infected. In our experience relapses are frequent, but with care in feeding and the administration of some chemical inimical to the development of the giardiae very satisfactory results are obtained and the ill effect on digestion and growth is minimized.

Treatment.—All of our cases were given bismuth salicylate in doses of 2 to 10 grains, according to age, three or four times a day. This is the drug especially recommended by Wenyon. The medicine was given continuously for a week or more, then discontinued for a week. By repeated use of this drug during several months all the cases under observation except one (*Case 1*) became free from giardial cysts. Bismuth salicylate even in large doses failed to rid the intestinal canal of the parasite, although the stools became hard and dry.

We succeeded in this case by the administration of stovarsol, at first giving only one grain twice a day, then increasing the dose to four grains daily. No cysts were found a month or more after this treatment had been instituted. I will admit that these cases have not been under observation for a sufficient length of time for positive conclusions as to the efficiency of treatment to be drawn. All writers are in accord on the difficulty of removing this intestinal parasite permanently. Yet the repeated administration of bismuth salicylate over several periods of a week or more, certainly improved the clinical condition, checked the excessive fermentation and permitted normal development.

I am fully aware that proof of the pathogenic influence of these flagellates in older children is inconclusive. The child or adult generally adjusts himself as a host to this parasite, but the infant with its short intestinal canal and its digestive apparatus always working at its highest capacity, is appreciably disturbed by infestation with the giardiae. This is my conclusion after a study of ten cases.

SUMMARY

The *Giardia intestinalis* is frequently found in the feces of children and in infants. In older children its presence is not always accompanied by digestive or nutritional disturbance.

In infants, however, the intestinal infestation of this parasite produces an increased intestinal peristalsis, a lessening of food absorption, and a consequent retardation in growth.

The administration of bismuth salicylate several times a day for a week, repeated several times during six months or more is effective in improving the clinical condition and may remove the parasite permanently.

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DISCUSSION (*Abstract*)

Dr. L. R. DeBuys, New Orleans, La.—Any organism which does not belong normally to an individual, is worthy of interest and study. The organism that Dr. Zahorsky brings to our attention today is one which is frequently found by accident. It has been variously named and is known as *Giardia intestinalis, Lamblia intestinalis, Megastoma intestinalis* and *Megastoma entericum.* The first to describe this organism was Lambl in 1854; he found it in the mucous evacuation of Russian children. The organism lives in the intestines of rats and mice; it has also been found in the rabbit, cat, dog, sheep and man. Rats and mice are to be regarded with suspicion as possible disseminators of

the infection, their feces infecting food and water. Infection in man is through the ingestion of the encysted form.

Whether the organism is pathogenic to man has been a matter of some difference of opinion. Certainly it is not fatal, and for that reason pathological studies have not been possible. Of all the protozoa occurring in the intestinal canal of man other than the *Entameba histolytica, Entameba coli,* and *Balantidium coli,* the *Lamblia intestinalis* has the best claim to be regarded as of pathological importance. Griffith in 106 patients in the Out-Patient Department and wards of the Children's Hospital of Philadelphia, found this organism in 22.6 per cent. Maxy, in 89 children, found its incidence to be 25 per cent. Undoubtedly the organisms may be present without causing any symptoms, although they are believed by many to be responsible for some forms of diarrhea, as in the cases of Dr. Zahorsky. This diarrhea may be severe and it may be accompanied with the pain which he describes. The stools contain a great deal of mucus, and usually many of the active forms are seen. The habitat of the flagellates is chiefly in the duodenum. They may be found in fewer numbers as far down the intestinal tract as the ileo-cecal valve. It is believed that the flagellates attach themselves to the mucous membrane of the intestine by means of their suckers and pass a great part of their existence there. In their natural surroundings they are not actively motile. Encystment is a common feature of this parasite.

With the more recent activities in gastro-enterology the organism has received more recognition as an invader of the duodenum and has been seen in its active state and vegetative forms there in the fresh duodenal contents; and also giardia have been recovered from the gall bladder bile at operation. It has been my custom for many years to examine the stools of all children who come to me at their primary examination because we have many cases from the tropics. In this way are found infections that might otherwise be overlooked. We are using the staining methods used by Dr. Zahorsky.

In a series of four cases seen by me several years ago, one was brought for an intestinal disturbance and three for a general investigation. Three had a malnutrition and four a chronic intestinal indigestion. All had a secondary anemia. The eosinophil counts were 2 per cent, 3.5 per cent, 4 per cent and 6 per cent. Three cases were discovered on routine examination of the feces. One case also had an infection of *Tricocephalus dispar* and another had an associated hookworm infection. This latter one was the only one with an eosinophilia.

In the treatment of these cases the gastro-enterologist has used biliary drainage and irrigation of the duodenum by duodenal tube. Medicines have been given by every route but nothing has yet worked satisfactorily in clearing the infection. One of my patients was apparently well after a period of self-starvation. The organism was not found again in the stools on many subsequent examinations over a prolonged period. Various arsenical preparations have been used, including stovarsol, and arsphenamin with no uniform results. Thus far no specific drug or method of treatment has been found.

Dr. Zahorsky (closing).—I can not insist that this protozoon is pathogenic. Still, in the few cases that have been studied, disease and the organism were certainly co-existent.

The condition needs careful study and by microscopic examination of every stool. When you see these bright, shining bodies you can recognize the *Giardia intestinalis.* It deserves our attention, especially in the South.

PNEUMOTHORAX THERAPY*

A CONSIDERATION OF ITS VALUE AND APPARENT NEGLECT

By CHARLES HARTWELL COCKE, M.D.,
Asheville, N. C.

With a decreasing mortality rate that has demoted tuberculosis from captain of the cohorts to fifth place in the rank of the causes of death, it seems well to pause and consider the modalities which have stood the test of time in achieving this happy reduction. It will be manifestly impossible to mention even, much less discuss and assay, here all of the many factors contributing to this lowered rate. Such a study might with advantage occupy an entire session, for it is the story of the hygienic and socio-economic progress of the past quarter of a century. The remarks that follow, however, are not based upon the assumption that this remarkable drop in the mortality from tuberculosis is the result of progress alone in the treatment of the disease, encouraging as this progress is. Since however lowered and lowering mortality rates must bear some definite relation to successful therapy, my remarks are addressed to a phase of successful treatment which has not had the continuous acknowledgement before this body that it deserves. I am discussing only one of the proven methods of treatment, which a widening experience and the passage of time, allowing opportunity for observation of its efficacy, attest as the one outstanding contribution to tuberculosis therapy within the past twenty-five years. I refer to collapse therapy and shall discuss only one phase of this, the induction of artificial pneumothorax.

REST-FRESH AIR CURE

Within the past quarter of a century four general methods of treating pulmonary tuberculosis have had sufficient exploitation to make judgment upon their value reasonably safe. The first, the fresh air rest cure method, is still our sheet anchor. Undoubtedly expanding knowledge of tuberculosis especially as a result of the general use of the x-ray, and greater facilities for the rest and fresh air method, have added much

*Read in Section on Medicine, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

to the refinement of its use; but its principles are fundamental and I agree with Edouard Rist, that granted the happy day when some biological product, (or vaccine) or chemical compound may be found to act as a specific, there will still be the necessity of relying upon the essentials of rest, fresh air and nourishing supportive diet. There are still certain cases of tuberculosis which despite the best of nursing care, the most loyal and painstaking cooperation on the part of the patient, the most thorough and detailed instruction upon the part of the physician under conditions which approach if they never attain the ideal, still slowly progress and extend and inevitably go on to a fatal result, unless more and better rest is afforded the patient than he gets from lying in bed: that is, unless the burden of respiration is removed from an already overtaxed lung and the absorption of tuberculotoxin is taken from a body whose reactive capabilities are already swamped. In other words, rest, though our only known specific in tuberculosis, does not have its fullest chance if we limit its meaning to bed rest in the open air.

IMMUNOLOGICAL

The immunological, or second method, has been, if not a total, almost a complete failure. Tuberculin is now rarely administered as a therapeutic agent, for the disappointments following its use in countless thousands of cases and in its numerous and various forms must have saddened practically all students of tuberculosis. The brief day of the vaccine had its golden apogee stimulated by the zeal of the biologic products manufacturers, and died an unmourned death. Since I reported some observations before this Section on this subject at the meeting in Chattanooga,[1] I wish to stress again the futility of such measures. And since active immunization of a serum producing animal has never been successfully accomplished, it naturally follows that passive immunization by serum therapy has been a chimera. If our experience with tuberculin as a cure has been a total disappointment it has at least taught us a very healthy respect for its potentialities for danger. If immunological treatment has played any significant part in our advancing successful treatment of tuberculosis, I am unaware of it. This statement is not meant in any sense as a criticism of the brilliant claims of Calmette with his B. C. G. immunization experiments. It seems worth while to state, however, that Calmette's work is still unconfirmed sufficiently for complete acceptance, and further to add that so far the method of immunization is applicable only to the newborn who have not yet come in contact with the ubiquitous tubercle bacillus, and is not at all suitable as a therapeutic measure in those who are known to be affected.

CHEMOTHERAPY

The third method, chemotherapy, has achieved added interest of late by reason of Moellgarrd's announcement of sanocrysin, backed as it was by the commendation of such high authorities as Thornwald Madsen and Knud Faber. And yet, as reports come in from all countries from competent workers in laboratories and from experienced observers in tuberculosis therapy, one is inevitably forced to the conclusion that gold and sodium thiosulphate is powerfully toxic to the tuberculous, that the drug has no favorable influence on tuberculosis of the lymph nodes, of the skin, of bones and joints, and that while in certain selected pulmonary cases under carefully guarded dosage it may apparently affect the outcome, its effects in tuberculosis can by no means be compared with those of arsphenamin in syphilis. Only an open mind as to its efficacy is the proper attitude. Until further study and information have settled many points, we must give it the verdict of "not proven" which also applies to other chemo-therapeutic agents.

ARTIFICIAL PNEUMOTHORAX

The last modality, collapse therapy, unless otherwise specified this term is used to signify artificial pneumothorax and does not embrace such surgical procedures as thoracoplasty and phrenicotomy, a mechanical measure, while recent in its general use has an acceptable, if not venerable ancestry. Artificial pneumothorax was first suggested by James Carson about 1819, (although Krause[2] tells of its inclusion in the writings of Hipprocrates). Though it was used in a few cases by John B. Murphy, the surgeon of Chicago, 1898, its wide acceptance today is undoubtedly due to the vision, faith, and unremitting toil of Forlanini of Pavia whose first publication on the subject dates from 1894. Honorable mention should also be made of the work of Dr. Mary Lapham, of Highlands, North Carolina, whose publications on the subject were among the earliest of American writers.

Pneumothorax therapy is the one outstanding brilliant advance in the treatment of tuberculosis achieved within the past twenty-five years, however brilliant may be the future of surgical collapse therapy. It has become a routine measure in every well conducted sanatorium

and is in constant daily use in the hands of every well trained tuberculosis therapist. It has in many instances so completely revolutionized prognosis that enthusiasm for its benefits, in certain apparently hopeless cases, is restrained with difficulty. Its wide-spread use is readily explainable in institutions and hospitals for the care of the tuberculous, where aseptic technic and the general use of the x-ray have materially lessened its dangers and enlarged its sphere of usefulness.

However, if one is to evaluate properly its usefulness, a brief consideration of its applicability is in order. At first it was commonly assumed that it could be used only in a case in which the involvement was practically unilateral and yet one where there was an advancing lesion, or at least a lesion little modified by prolonged conservative measures. Naturally, even before the general use of the x-ray, the impossibility of finding a strictly unilateral case pathologically (although the unilateral case clinically is less rare) had become quite apparent. With the general use of the x-ray this has inevitably become quite rare. But by this same token its ever widening sphere of usefulness has been achieved; for the x-ray has taught us much of the state and age, and consequent activity of the foci, physical examination of which left much to be desired in the matter of exact knowledge. Briefly the following groups of conditions may be considered as suitable ones for the procedure:

(1) Unilateral progressive tuberculosis unmodified by conservative measures, whether an acute, infiltrating, cheesy, pneumonic process or a chronic fibro-caseous process with or without destruction and cavitation.

(2) For the replacement of extensive pleural exudate.

(3) For the control of hemorrhage or too often repeated hemorrhages with the consequent danger of dissemination therefrom.

(4) For the continuance of spontaneous pneumothorax in the effort to allow the lung to heal, to prevent pleural infection and a repetition of the accident as well as to allow amelioration of the original pathological condition.

(5) And lastly, as a palliative measure in certain cases for the amelioration of distressing cough and expectoration.

This surely represents as unfavorable a group as one could imagine. A rough estimate is that these cases represent about 10 per cent of the patients in a health resort, and yet it is in just these desperate cases that the method has achieved its most brilliant results.

THE CONTRALATERAL LUNG

Naturally the final decision as to the advisability of attempting the method is based upon the supposed integrity of the lung which is called upon to assume the entire burden of respiration. It is here that the most meticulous study, the most careful correlation of physical, laboratory and roentgenologic observations is a *sine qua non.* And yet, increasing experience has broadened wonderfully the type of cases susceptible of successful treatment by this method. It is not the purpose of this paper to go into details of suitable cases and their proper method of selection, which would require a separate paper, but it is desired to point out that the former too conservative method of using pneumothorax only in a case that showed no demonstrable lesion in the opposite lung was denying an opportunity for help to many cases that are now successfully so treated. (We now practice the method bilaterally in certain carefully studied and more carefully treated cases.) I find in my own practice an inevitable tendency not to wait quite so long as formerly before deciding on pneumothorax. The longer one delays the greater the probability of adherent pleurisy and consequent inability to effect a working pneumothorax, and the almost inevitable certainty of great destruction of tissue. The method is of course not begun at the outset of treatment, but only after conservative routine rest is thoroughly tested and has proven a failure. Since tuberculous lesions in the two sides of the chest are rarely of the same kind, age, and extent pathologically, it is a fair assumption that in practically all cases of very far advanced tuberculosis, there was a time when artificial pneumothorax might well have saved the patient's life and even restored him to economic efficiency. I state this rather categorically although I am well aware of the fact that determining just when this time is may be fraught with insuperable difficulties. Some cases seem in an incredibly short time to become almost hopelessly advanced bilateral ones, even before the patient has any reasonable comprehension of his illness. I do not consider it a fair test to relegate its use to those cases in which it seems to be considered as a last refuge: all too frequently in these the last chance is the lost chance. It is to the prevention of the lost chance to which I ask your attention.

But lest you mistake my enthusiasm for general advocacy, again I would emphasize certain things: first, that pneumothorax does not in

any sense replace conservative rest treatment, and every case should have the benefit of careful observation before the method is undertaken, and likewise afterwards.

Meticulous attention to bed rest is very important in the early stages of pneumothorax therapy, and pneumothorax by no means relieves the patient of the necessity of careful rest for quite a period after the induction is commenced. And while it is conceivable and quite probable that every advanced case might have been helped by it at some time, this by no means is an open invitation to try it on all such cases. Again, while it calls for skill and judgment, as well as experience in its use, it is no more difficult a procedure, once it is successfully begun, than thoracentesis, and should be possible in the hands of any man who feels competent to tap a chest. Lastly, it has its definite limitations, many contra-indications, numerous complications, and distressing failures.

RESULTS

What of its results? While statistics are available in series that run from a few score to thousands, it is not necessary to review these here. Any competent tuberculosis practitioner should be able to report successes to the point of restoration to ordinary life and economic independence varying from 30 to 50 per cent, with further amelioration of symptoms and prolongation of life in another 15 to 20 per cent, in cases that would usually have gone on to fatal termination. In the fall of 1926 Edouard Rist[3] gave his results before the Washington meeting of the National Tuberculosis Association. He showed that of 759 cases of tuberculosis treated by artificial pneumothorax the healed and clinically well group represented 52 per cent, with 48 per cent worse or deceased, while of 213 patients who refused it and thus became a control group, the worse and deceased group is represented by the figure of 82 per cent. I refrain from detailed statistics as the factors which modify proper comparison, for example, the types of cases selected, earliness of institution of the method, length of time used, complications, tuberculous or otherwise, skill and experience of the operator, degree of collapse, and control of the patient during treatment, are numerous. I wish to emphasize particularly the remarkable opportunity the method offers, now that it can be checked by the fluoroscope and films.

What of its general use by competent internists? As I said earlier, while the method presents no greater difficulties than thoracentesis and the equipment is only a slightly more elaborate and expensive one than is necessary for taking fluid out of the chest, inquiry among prominent internists of some of the leading cities of the South shows a remarkable paucity of men equipped for and capable of doing this work. I could learn of only two men in the fine medical center of Memphis; none in Birmingham, save the Medical Superintendent of the Municipal and County Tuberculosis Sanatorium; only three in Atlanta and four in New Orleans, equipped for this work. What prompted this paper was the shock of hearing at the recent meeting of the National Tuberculosis Association in Indianapolis, the remark by the medical superintendent of one of the largest and best equipped tuberculosis sanatoria in the South that he "didn't use pneumothorax because it was no good;" and a further reason was the really difficult time one has in directing for refills a patient who must return home though still in need of compression. As you are all aware, the economic condition of the patient about as frequently determines his successful fight with tuberculosis as does his physical. After some months of sucessful pneumothorax therapy, not a few patients might be allowed to return home from health resorts to continue the collapse, were properly trained physicians at hand to keep up the treatment intelligently and successfully. This would be the means of invaluable economic as well as medical relief to many poor people who now return home too soon, or fail of the benefits of pneumothorax therapy because of lack of adequate opportunity for treatment. The human, as well as the economic phases of tuberculosis, demand that this method should be more generally available to the patient. And yet a search through the programs of this Association revealed the startling information that the subject has not been discussed in this Secton since 1916, despite its enormous medical and economic importance.

In this connection I am tempted to quote an interesting experiment being tried out in Detroit. Realizing that the tuberculosis problem is an economic one, as well as a physical and pathological one, a certain group of physicians in Detroit with a large clientele of tuberculous patients who were unable either to go away for treatment or even to stop work, has instituted artificial pneumothorax in this group of patients as soon as the diagnosis was made. The patients returned immediately to work, or rather never ceased working. It is not claimed by these men that this is an ideal procedure. It is only an earnest effort to solve a problem otherwise in-

soluble, and does this by not changing the economic status of the patient. It is too soon to evaluate the results of this experiment. I mention it to emphasize the enormous economic potentialities of pneumothorax therapy. The burden of proof is entirely upon those who deny the efficacy of pneumothorax.

In conclusion I wish to emphasize:

(1) That artificial pneumothorax therapy is the one outstanding contribution to tuberculosis therapy within the past twenty-five years.

(2) That its value, while limited, is proven from every aspect, human, physical, and economic.

(3) That it is not beyond the possibilities of any first class internist who will take the time to familiarize himself with it.

(4) That internists of the South in sufficient numbers have not qualified themselves for it.

(5) That such a condition is a reproach to the second largest medical association in the country, and that you gentlemen of this Section are the ones to correct it.

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Discussion follows paper of Dr. Laws, page 605.

ADMINISTRATION OF ARTIFICIAL PNEUMOTHORAX*

By J. W. Laws, M.D.,
El Paso, Texas.

Before enumerating the points to be considered in the administration of artificial pneumothorax, a brief statement of what pneumothorax is and how it came into use will possibly give a clearer idea of how its present day use has become an established therapeutic measure of value.

Pneumothorax as applied today consists of the introduction of air into the pleural space, thereby placing a pneumatic splint around the affected lung, compressing it and giving the lung more or less physiological rest from function.

Carson, a physiologist, in 1819-1821, recommended such a procedure on theoretical grounds. Houghton, in 1832 and Stokes, in 1837, reported cases where spontaneous collapse was followed by marked improvement in patients suffering from pulmonary tuberculosis. Fornalini, in 1888, began to put to a practical test the use of artificial pneumothorax, reporting some of his cases in 1894 and 1895. John B. Murphy, in 1898, advocated its use on surgical principles and himself treated five cases. Brauer, in 1905 and 1906, reported his method of inducing artificial pneumothorax, and the use of the water manometer. From 1905 on up to the present time experience has been accumulating in the use of pneumothorax until today it is an established remedy of value in certain types of pulmonary tuberculosis.

There are certain types of cases, certain indications and contra-indications and certain complications that must be considered in the administration of pneumothorax. In order to facilitate the discussion a brief tabulation follows:

I. CASES SELECTED FOR ARTIFICIAL PNEUMOTHORAX

1. Moderately advanced cases.
2. Far advanced cases.
3. Cases showing progression of the tuberculous disease where other measures of rest and hygienic living have failed.
4. In moderately advanced and far advanced cases necessitating prompt return to earning capacity, pneumothorax should be used.

II. INDICATIONS FOR ARTIFICIAL PNEUMOTHORAX

1. Severe hemorrhages of exsanguinating type.
2. Uncontrollable recurring hemorrhages.
3. Partial replacement with air in cases where symptoms necessitate withdrawal of pleural effusion.
4. Cavitation that progresses.
5. Unilateral ulcerative cases with toxic symptoms not improved by rest cure.
6. Pneumothorax over worse lung in bilateral cases unimproved by other treatment.
7. Solely as a palliative measure to relieve toxic symptoms and render the patient's condition more comfortable.

III. CONTRA-INDICATIONS

1. Severe tuberculous entero-colitis.
2. Acute ulcerative laryngeal tuberculosis with dysphagia.
3. Widely disseminated miliary tuberculosis.
4. Minimal pulmonary tuberculosis.
5. Pulmonary tuberculosis with retrogressive symptoms and physical signs.

IV. ARTICLES REQUIRED FOR OPERATION OF PNEUMOTHORAX

1. Pneumothorax apparatus (Floyd-Robinson needles unnecessary).

*Read in Section on Medicine, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

2. Luer tuberculin syringe, 3 needles, 25 guage 1 inch, 20 guage 2 inch, 18 guage 2 1-2 inch, blunt point.
3. Novocain, 1 per cent tincture of iodin, alcohol.
4. Small sponges, gauze or cotton, a few towels.
5. Sharp pointed knife, Graefe's, for skin puncture.

V. AMOUNT AND FREQUENCY OF INFLATION

1. Small inflation under low pressure is ideal method.
2. Strongly positive inflations are dangerous and should only be used with a full knowledge of the pathological condition to be dealt with in the individual case.
3. First inflation only 300 c. c.
4. Second inflation usually 300 c. c. at 3 to 7-day intervals.
5. An average of 300 c. c. repeated every 5 to 7 days, the negative pressure gradually approaching neutral will frequently bring about satisfactory compression.
6. The amount of inflation and pressure will vary with the individual case.
7. Positive pressures are permissible with slowly yielding cavities where the displacement of the heart and the mediastinum is kept under frequent x-ray observation.
8. Hemorrhages are frequently controlled by small inflations (300 to 400 c. c.) but sometime require as much as 750 to 1500 c. c. at one operation. Repeated refills at frequent intervals is the wisest procedure, being governed by the requirements of the individual case.

VI. COMPLICATIONS

1. Air embolism.
2. Spontaneous pneumothorax.
3. Pleural exudates.
4. Empyemas with or without permanent bronchial fistula.
5. Development of progressive activity in contra-lateral lung.

VII. SUCCESS OR FAILURE OF PNEUMOTHORAX INFLUENCED

1. By complete obliteration of pleural space by adhesions.
2. By partial obliteration of pleural space by adhesions.
3. By length of time pneumothorax is administered.
4. By the complications in Table VI.

VIII. DURATION OF PNEUMOTHORAX TREATMENT

1. Collapse should be kept up as long as the patient is doing well.
2. After a period of years the lung may be allowed to re-expand under x-ray observation, observing any evidence of returning activity.
3. Discontinuance may be necessitated by progressive active disease in the contra-lateral lung and other complications in Table VI.
4. Discontinuance may be necessitated by formation of pleural adhesions.

DISCUSSION

Tables I, II, III.—In considering the advisability of administering artificial pneumothorax the physician will often find it difficult to place a given case of tuberculosis into a particular classification of indications and contra-indications. In such a case instead of postponing the administration of pneumothorax and using it as a last resort only, it is well to make up one's mind whether or not the case shows progressive activity and involvement, or retrogressive activity and involvement. It is necessary to consider the history of the case, weigh the physical and x-ray findings of the chest, and the laboratory findings. If the patient has been given rest in bed, hygienic and dietetic measures, a reasonable trial and the case shows progressive activity, then with the above classifications in mind, pneumothorax will be urged.

The profession and public must be brought to the realization of the fact that cases of pulmonary tuberculosis should have periodical examinations and medical supervision, otherwise so insidious a disease as tuberculosis will never be treated properly.

The family physician of necessity has to treat tuberculosis in the patient's home, but in this day and time he is usually situated near his state sanatorium or some medical center where consultation with a physician experienced in the treatment of tuberculosis can be had, and such counsel should be sought.

Cases of tuberculosis often wander around in the Southwest without medical examination and supervision, often actually having been advised by their physician at home to stay away from physicians, until pneumothorax can be tried only as a last resort.

Hemorrhage cases precipitate the use of pneumothorax, and emphasize the fact that the case would have drifted for months, except for this emergency.

There is a prevalent belief that pneumothorax should not be used unless one lung is absolutely free of trouble. Experience has taught, however, that the disease in the less affected lung often improves as the result of the gradual compression of the worse lung, probably due to the reduction of toxemia from the badly affected lung and the consequent improvement in the patient's general condition. In other words the lessening of toxic absorption resulting from partial compression of the badly affected lung more than counter-balances the extra functional work thrown on the less affected lung.

OPERATION OF ARTIFICIAL PNEUMOTHORAX

Table IV.—The technic of the operation for the administration of artificial pneumothorax should be as simple as possible, always bearing in mind the principles of asepsis and antisepsis. Particular care should be taken not to touch the needle to bedding, clothing, or anything that will carry infection. The operation may be done at the bedside of the patient in the home, at the physician's office, or in the operating room in the hospital or sanatorium. One should be sure that the apparatus for giving pneumothorax is not leaky and be sure of a good manometer oscillation before administering pneumothorax.

Occasionally the needle will be introduced as usual for a refill but there will be no manometer response. The needle may be blocked by a small blood clot, or there may be some pleural exudate filling the needle, or the rubber tubing leading to the apparatus may be kinked or leaking. A little investigation will soon discover the cause of trouble.

In administering refills, approximately the same area and intercostal space is used for needle puncture of successive inflations and occasionally it will happen that when the needle is introduced in the same place as in former inflations there will be no manometer response, or possibly only a slight fluctuation, and if the air is turned on the patient will complain of sharp pain and the manometer will quickly show a positive pressure. Removal and reintroduction of the needle in another intercostal space a short distance away will give a good manometer reading and the refill can be given without difficulty. I am inclined to believe the explanation of this phenomenon is due to thickening of the pleura around the area of previous needle punctures, or to pleural adhesions of the lung at that point, if the intervals of refills have been several weeks apart.

It sometimes also happens that when an attempt is made at the time for a successive refill, it will be found that the lung has re-expanded and all the former air in the pleural sac has escaped and no manometer reading is obtainable. X-ray findings will confirm the absence of air where formerly compression has been taking place. This condition happens, why, I do not know and can only surmise that the air has filtered out through some emphysematous bleb or thin cavity wall.

COMPLICATIONS

In air embolism, lower the patient's head, use artificial respiration, and administer stimulants.

Development of progressive activity in the contra-lateral lung, is of course an indication to allow partial or complete expansion of the compressed lung. After partial or complete expansion of the compressed lung it may be necessary to attempt gradual compression of the former contra-lateral lung, depending upon indications in the individual patient.

Occasionally the patient reports following the last refill that he "got a kick from that last shot of gas," some aching, a little fever and some soreness over the lower portion of the thorax on the compressed side. This should always be considered as an indication that probably the pleura is inflamed and that there may be some exudate, that the inflation was possibly too large, and before giving another refill the patient should be fluoroscoped. No refill should be given until after the acute symptoms have subsided. Where pleural exudates form, it is well to keep the patient under observation until after the acuteness of symptoms has subsided, and the intervals of refills should be lengthened, governed by the symptoms and findings of the individual case.

In the management of pleural exudates that increase to the point of gradually filling the pleural space, it will be found that the exudate, if unaccompanied by symptoms, will compress the lung satisfactorily and should be left alone and the refills deferred. However, such patients should be observed at regular intervals under the fluoroscope, for such effusions often absorb rapidly, particularly when the patient is taking ultra-violet radiation, and the lung may re-expand under a small amount of fluid and become adherent, particularly in the region of the costo-phrenic angle, and thereby complicate the resumption of the refills. Refills should, of course, be spaced so as to keep the lung under adequate compression. Mercurochrome, gentian violet and other dyes have been used where the exudate became sero-purulent or purulent, but the writer is not convinced that they are of any more value than is afforded by aspiration of fluid and air replacement.

In spontaneous collapse morphine and atropine are often given to relieve the pain and distress, and the intrathoracic pressure is overlooked. Where there is a valve-like action of the opening there is usually high intra-thoracic pressure. A needle should be introduced, a manometer reading made and air withdrawn until the pressure becomes neutral. If there is a recurring pressure and the symptoms are distressing it may be necessary to introduce and leave between the

intercostal space a cannula to maintain a neutral pressure.

Empyema following spontaneous collapse, where the bronchial fistula fails to close, has been most unsatisfactory to treat. Resection of rib and drainage is unsatisfactory because a large pleural cavity is left open, with no expectation that the lung will re-expand to fill the space. In a limited number of such cases phrenico-exeresis has been done followed by thorocoplasty. However, the results have been unsatisfactory in so far as our limited experience has gone, possibly due to the weakened condition of the patients upon whom operation has been performed. In a few cases the cutting of and extracting all of the phrenic nerve that is possible to bring away (phrenico-exeresis) without the operation of thoracoplasty has been done to stop the piston-like action of the diaphragm. This operation adds to the comfort of the patient and is of value. Discussion of the best posture and medical treatment of such patients, as well as the surgical treatment will be appreciated.

SUMMARY

(1) Artificial pneumothorax is a remedy of proven value.

(2) The operation is a simple one, but like all operations, even minor ones, certain points have to be considered that for the time being are often confusing.

(3) Pheumothorax, contrary to current belief, is not contra-indicated in all cases of bilateral involvement.

(4) Pneumothorax used as a last resort usually means negligent delay on the part of the patient or physician.

(5) Spontaneous collapse, pleural exudates and empyemas occur not infrequently where the operation for artificial pneumothorax has not been attempted. These complications should not deter the patient or the physician from the operation when it is indicated.

DISCUSSION (*Abstract*)

Papers of Dr. Cocke and Dr. Laws.

Dr. L. J. Moorman, Oklahoma City, Okla.—In discussing Dr. Cocke's paper I shall try to limit my remarks to his suggestion that certain internists in various sections of the country prepare themselves for the administration of artificial pneumothorax. There was a time when we had to go to a little out-of-the-way village in Italy to gain knowledge of artificial pneumothorax. Dr. Cocke is now recommending that artificial pneumothorax cases be turned over to the internist, especially for refills. Since Forlanini played the role of pioneer with reference to this therapeutic procedure, many obstacles have been removed, paths have been smoothed and guide posts have been planted.

There was a time in my own State when artificial pneumothorax was given only in my own city and often on account of economic conditions I have had patients go home and then make the long trip for refills. Now in the State of Oklahoma there are five places where physicians are giving artificial pneumothorax and it is a great help to the tuberculosis specialists, also to the patients from an economic standpoint, and to the physicians in the localities where they have prepared themselves for this work. It increases the interest in tuberculosis and will lead to an earlier solution of the tuberculosis problem.

In spite of the fact that the paths have been smoothed, there are yet many hazards, as Dr. Laws has pointed out, especially in determining the amount and frequency of fillings and there are various complications. Those of you who may be interested in this subject should study it thoroughly. The apparatus and the technic are simple. Anybody can give a refill, but knowledge of the disease and of the possible complications is essential, if artificial pneumothorax is to be carried on to the greatest advantage to the patient.

Dr. Virgil Simpson, Louisville, Ky.—It is quite true that Forlanini in his pioneer work without the aid of x-ray study established a practice which is of unquestionable benefit to a number of tuberculous patients. But it is important that the men who now decide to undertake this thing should bear in mind that physical examination alone is not sufficient for a satisfactory carrying on of the procedure. Pleurisy is a very common accompaniment of tuberculosis. With this, definite adhesions necessarily take place. The adhesions are frequently multiple and they not infrequently form compartments. The introduction of a needle into one of these will result in a rapid rise of the manometer reading. One might think, perhaps, with inexperience that a point had been reached justifying discontinuance of the procedure, whereas a great part of the lung has not been compressed. Checking the procedure with the fluoroscope is a material help.

If he has x-ray assistance there is no reason why any competent internist should not undertake this work.

I am not yet a convert to the idea that a bilateral involvement of the lung, active on both sides, is not a contra-indication for the use of pneumothorax. If rest is of the greatest importance to a lung in the management of tuberculosis, and it is certainly conceded to be, it does not seem logical that to collapse one lung and allow the other lung which is actively diseased to undertake the burden of supplying the oxygen for the entire system is fair treatment. I have always been exceedingly doubtful about artificial pneumothorax when I know that both lungs are actively involved.

Dr. Roy Goggans, Dallas, Texas.—It is easy to become an enthusiast in the use of this treatment for pulmonary tuberculosis, because there is nothing that can accomplish such results in the suitable case in a short period of time. On the other hand it is very easy to have our enthusiasm upset by the immediate and late complications which sometimes develop in the course of artificial pneumothorax. One of the dangers, that was not forcibly emphasized in either paper is pleural shock. Some very good authorities deny that this complication exists, but few men in the country today who are giving artificial pneumothorax have failed to encounter this clinical condition, regardless of what it may be due to. In the majority of cases it is due

to carelessness in making the puncture, and lack of skill in the use of the local anesthetic.

Dr. Laws has mentioned two of the contra-indications that are usually given in all text-books and papers on this subject: they are acute tuberculous laryngitis and tuberculous entero-colitis. I believe that the time has arrived when we should not consider either one of these as a contra-indication for pneumothorax if there is a unilateral, or even a bilateral, pulmonary condition that might be benefited by it. The treatment itself does no harm, if carefully used, in either of these complications, and frequently is of definite benefit to a patient. We know that in the majority of cases both of these complications are secondary to the lung condition; and if we can succeed in cutting the patient's toxemia down with artificial pneumothorax, then his throat or his bowel complication may have a chance to improve as the result of raising his general resistance. Why not give him the benefit of the doubt?

Dr. E. C. Thrash, Atlanta, Ga.—I began this work seventeen years ago and think I have had practically all the complications that we know of. In regard to pleural shock, I doubt that there is any such thing. It is a good alibi, but our casualties come from two outstanding things: one is air embolism and the other is the effect that is produced upon the heart by displacement, especially to the right side, which is a very good point to bear in mind in giving pneumothorax. Some of my most distressing results have been due to the effect produced upon the heart by this pressure and that is what is usually called pleural shock.

A fixed mediastinum is best in these cases. Where there is a mediastinitis with thickening of the mediastinal wall and adhesions it remains standing and you get good pressure. You have a parietal wall on the one side and a mediastinal thickening on the other, which will take better care of the heart. In viewing my films I am always glad to see that I have strong evidence of a mediastinitis, for I feel that I have a substantial wall against which I will get pressure. But do not forget that you have a heart lying between the two lungs, and you are pressing just as hard upon that as you are upon the lung structure. If you introduce a little too much air you will find that the heart has picked up from 80 or 90 to 120. This is a danger signal and you should be more careful next time and let your manometer reading be lower than it was before. I have been quite as much worried over the heart problems as over the other condition.

Dr. W. L. Dunn, Asheville, N. C.—Pleural shock, air embolism and the other so-called complications of artificial pneumothorax are not frequently seen. Of course, they are very serious when they do occur, but they occur with such infrequency that I believe the real tragedy is the reaction of some focus in the contra-lateral lung. When we induce artificial pneumothorax in the tuberculous patient we are putting the bad lung "to bed" and the good lung "on exercise." If we will always keep that in mind it will also emphasize a very important thing to the man who is in general practice, who does only the occasional refill, that the technic of collapse is unimportant as compared with the careful observation of the contra-lateral lung.

I beg of those who expect to undertake artificial pneumothorax without a wide experience in its use, to keep constantly in mind that the real tragedy is reactivation of any focus in the contra-lateral lung. Also, the physical signs which we expect to find ordinarily in a normal lung are more or less changed by the compensatory breathing which takes place after the collapse of the other lung.

Dr. R. E. Flack, Memphis, Tenn.—When pneumothorax was first introduced, the cases selected for its administration were usually far advanced and many times hopeless; or it was given to control hemorrhage. After years of experience and more careful study of various types of tuberculosis, physicians are administering pneumothorax in earlier cases with excellent results. Every case of tuberculosis should be studied with reference to pneumothorax, even at the first examination. I predict that pneumothorax will be used in early destructive lesions in many cases and they will not be allowed to develop into the more advanced stage.

A physician who attempts the induction of pneumothorax should not only be trained in its technic but should have a full knowledge of its dangers. Experience alone will give the operator surgical judgment to determine when it is impossible to find a free space in the pleural cavity and to be able to adjust the desired compression. Refills are very simple and can be done by any one who possesses an apparatus. In addition to training, a physician should have the aid of a fluoroscope and x-ray films.

Dr. W. E. Vest, Huntington, W. Va.—What are the indications for pneumothorax in lung abscess, and under what circumstances is it advised? In what percentage of cases of tuberculosis is pneumothorax done?

Dr. Charles L. Minor, Asheville, N. C.—There is of course a large number of cases in which the adhesions make pneumothorax impossible. But here fortunately we can fall back on thoracoplasty. Furthermore, Dr. Matson of Portland, Oregon, is getting very good results with Jacobaeus cautery of the adhesions, which will greatly enlarge the number of cases that we can cure, but this latter method is, at present at least, a very special thing and takes special technic and training.

Any intelligent physician can learn the technic but the technic is only part. He has got to understand the disease; when to increase pressure; when to decrease it; when to go slow; when to go fast, and therefore, I do not think it should ever be used by the general practitioner, but rather that men who understand the method should be scattered here and there through the state.

As regards bilateral pneumothorax, we are using it in certain carefully selected cases and have done no harm with it at least. I trust we have done good, but it is too soon to speak.

Dr. Cocke (closing).—I would not have you feel I recommended the indiscriminate use of pneumothorax by the untrained and inexperienced. I prefaced my remarks by saying I was discussing the method only with reference to pulmonary tuberculosis. However, fluid occurs in between 50 and 60 per cent of cases so treated.

There are between a million and a million and a half cases of pulmonary tuberculosis in the United States not under the care of the tuberculosis expert or specialist. A very small proportion of these comes under the care of such men and that means that a large proportion of the cases is in the hands of the general practitioner or should be. What I want him to get from this discussion and paper is not necessarily that he must become an expert in tuberculosis or an expert operator with the pneumothorax apparatus, but that he must know something of the possibilities that this procedure offers. If he does not care to take the time and trou-

ble to equip himself to give his patient the benefit of this well proven method of arresting the disease, he can at least refer his patient to some one else. Rest, I mean body rest in addition to mechanical rest, is the *sine qua non*, but this method of treatment deserves more serious consideration than is frequently given it.

Dr. Laws (closing).—The majority of cases of lung abscess that I have seen have been of long standing with dense adhesions preventing the administration of pneumothorax and were per se surgical cases. Where it is possible to compress the lung and cavity area of abscess, artificial pneumothorax is of value.

The value of artificial pneumothorax in pulmonary tuberculosis is recognized by physicians who specialize in the treatment of pulmonary tuberculosis and its neglect is constantly emphasized by the case whose trouble has been allowed to progress until its attempted use is a last resort and often a failure.

Tuberculous patients should be under the supervision of a physician. The physician should at stated intervals correlate the patient's symptoms, physical signs of his chest, x-ray and laboratory findings. If the case is progressively active despite rest in bed and other constructive measures, artificial pneumothorax should be considered.

TUBERCULOSIS IN RELATION TO THE EYE*

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This subject readily lends itself to partisanship. So protean a disease as tuberculosis is always in the minds of some physicians and they suspect it in many varieties of case. Others associate the word tuberculosis with cough and emaciation and find it impossible to believe that an apparently vigorous man can have tuberculosis in any organ. The personal equation enters very largely into the interpretation of the results of such an agent as tuberculin, necessarily used in very small doses and continued over a long period of time. Therefore, a wide variety of opinion is heard in regard to the value of this method of diagnosis and treatment. The attempt will be made not to espouse a cause, but rather to present the known facts and probable theories in regard to ocular tuberculosis, and to suggest some practical questions for discussion.

There can be no doubt of the existence of tuberculous disease in the eye, human as well as animal.

As long ago as 1899, Fredrich and Nosske[1] produced tuberculosis in rabbits by injecting living bacilli into the arterial circulation. The ocular lesions were incidental to their main problem of generalized tuberculosis. Stock,[2] in 1903, experimented particularly with tuberculosis in the rabbit's eye by injecting bacilli into the ear veins. In these and subsequent experiments he observed tuberculosis in every structure of the eye except the lens, and even cataract was an end result in a severe case. Otori[3] found tubercle bacilli in the human retina. Ginsberg[4] found bacilli in sections from a tear sac, a scleral nodule, and two cases of intraocular tuberculosis with perforation. Bergmeister[5] found bacilli in a conglomerate tubercle of the choroid with detached retina. Bacilli have been found in conjunctival lesions frequently.

Inoculation experiments, while not uniformly successful, are positive often enough to prove that lesions regarded clinically as tuberculous in the human eye reproduce lesions accepted as tubercles in animal eyes.

For example, Lawford[6] placed tissue from an episcleral tubercle of a child's eye into the anterior chamber of a guinea pig, with the production of tuberculous iritis. Later the child developed tuberculous glands in the neck. Collomb[7] had an identical experience. Bruns[8] produced phlyctenules of the conjunctiva in rabbits after injection of bacilli into the common carotid artery. Amano[9] obtained phlyctenules by increasing the susceptibility of animals to tuberculosis. Haensell,[10] and later Panas and Vassaux,[11] produced corneal tuberculosis in the rabbit. Langhans[12] and Haensell[13] produced conjunctival tuberculosis in rabbits by instillation of tuberculous material into the conjunctival sac. H. Lagrange[14] accomplished the same result with emulsions of tubercle bacilli.

The literature of this subject is vast, as one can readily see by a glance at the index of successive volumes of the "Ophthalmic Year Book." The reports of Stock[2] deserve careful study, and particularly the extraordinarily complete and careful investigations of Finnoff,[15] which constitute one of the outstanding contributions of American ophthalmology to exact science.

He has demonstrated the occurrence of tuberculosis in all the ocular tissues of the rabbit, except the lens, by the hematogenous route, using first emulsions of living tubercle bacilli, and then of dead bacilli, injected into the common carotid artery. In the former group he obtained the severer lesions of the human type, and certain more rapidly destructive, and therefore dissimilar, lesions. The rabbits usually succumbed to generalized tuberculosis. In the latter group less severe lesions occurred from the effects of dead bacilli; the rabbits lived many months and the picture of human tuberculosis was reproduced. They were the well recognized histologic pictures seen in undoubted tuberculosis in human tissues.

One who is disposed to be skeptical of ocular tuberculosis should understand that this is not altogether a matter of clinical enthusiasm, but that many and rigid proofs of this disease are recorded. There may be debate as to some features of individual cases, but there can be no doubt that tuberculosis of the eye occurs.

The tissues adjacent to the eye show tubercu-

*Read in Section on Eye, Ear, Nose and Throat, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

losis as follows: the orbit may be the seat of a tuberculous mass arising from the sphenoid or ethmoid, as found by Lebenhart[16] in two children; optic atrophy and exophthalmos resulted. The lacrimal gland is rarely involved, but a few cases are recorded.

Abadie[17] made the first report in 1881; Plitt[18] was able to collect twenty-six cases in the literature in 1906, and Beauvieux and Pesme[19] reviewed the subject at length in 1922. It is very possible that the so-called Mikulicz' disease, in which the lacrimal gland is involved, is a tuberculous infection, as Napp[20] argued in 1907, and Fleischer[21] in 1910. The lids are affected by lupus extending from the adjacent skin. "Cold abscess" may occur subcutaneously in the lid. Tuberculous tarsitis has been described by several writers, especially Rollet.[22] H. Freidenwald[23] has recorded a case of chalazion with the typical histology of tubercle.

Conjunctival tuberculosis has received much attention in Europe, and the disease is classified systematically. Apparently there is more tuberculosis among the poor in European cities than in America.

Dimmer[24] speaks of five forms which tuberculosis assumes in the conjunctiva: (1) ulcers, with ragged edges and bacilli present; (2) trachoma-like granulations; (3) lupus conjunctivae; (4) cockscomb-like proliferations; (5) tumor-like pedunculated structures. He would make a sixth variety of Parinaud's conjunctivitis. This seems hardly permissible in view of the evidence which Verhoeff[25] has brought forward showing a leptothrix as the cause of this disease. The literature of this subject is abundant. Much attention has been given to the mode of infection, the primary lesion being found in the exposed surface, trauma facilitating the lodgement of bacteria, as Fuchs[26] has emphasized.

There is, on the contrary, much evidence for the secondary nature of conjunctival infection, the nasal mucous membrane, lacrimal sac, and blood stream being incriminated as sources of infection. The extension of palpebral infection is noted in the pre-auricular gland chiefly, in the adjacent skin and subcutaneous tissues, and in the bones (especially the malar).

The question of the relation of phlyctenules to tuberculosis is still under debate. These lesions are not tubercles histologically and they do not contain bacilli. Therefore, they cannot be regarded as tuberculous lesions in the strict sense. They do behave, however, in ways that suggest a relationship.

Bruns[8] produced phlyctenules in the rabbit's conjunctiva, but not in the cornea, by injecting an emulsion of dead bacilli into the common carotid artery. The greater incidence of the positive von Pirquet test in phlyctenular than in non-phlyctenular cases is suggestive. This is by no means conclusive, for the von Pirquet test is not of great value in adults and older children; but it cannot be dismissed as altogether useless. Wilder,[27] reporting for the Committee of the Ophthalmic Section of the American Medical Association appointed to study this question (1910), stated that 47 out of 57 cases of phlyctenulosis gave distinct evidence of tuberculosis, active or quiescent. Guillery[28] concluded from experiments that phlyctenules represent a tuberculo-toxic disease, without bacilli. Igersheimer and Prinz[29] called attention to the fact that phlyctenules showed no tendency to develop into genuine tuberculosis of the cornea, nor are they usually associated with other tuberculous processes in the eye. Eyre[30] believes that phlyctenules occur in eyes which are irritable because of some local condition in a person with low resistance, and any organism may then produce a lesion out of proportion to its accustomed virulence. Clinical experience indicates very forcibly that this affection is best treated as if the individual were tuberculous; the old term "scrofulous" well expresses the condition of the patient, though his ocular lesion is not a tubercle.

Rollet and Colrat[31] use a suggestive term throughout their recent work on ocular tuberculosis, "inflammatory tuberculosis," to describe the inflammatory reaction of various tissues to what they regard as a tuberculous toxin. Phlyctenulosis comes under this heading. The tuberculous etiology is indicated by tuberculous antecedents, glandular enlargements, cutaneous reaction to tuberculin, ophthalmo-reaction and animal experiment.

The cornea is involved in an interstitial lesion very similar to leutic keratitis. There is usually, however, a less uniform infiltration, more limited to one portion, beginning at the limbus. Tongue-like projections extend into the parenchyma. These lesions are usually associated with deep scleral involvement which extends into the iris root and pectinate ligament as well as the cornea; in addition, there are "mutton-fat" deposits in the deepest layers and on the posterior surface of the cornea, as Posey emphasized. Thus keratitis and iritis occur together.

Bach[32] made the following classification: (1) a parenchymatous type, due to the action of tuberculous toxins on the parenchyma; (2) a sclerosing type, due to tuberculosis of the pectinate ligament; (3) keratitis of the corneo-scleral margin unassociated with tuberculosis of the iris or pectinate ligament; (4) a corneal affection secondary to tuberculosis of the conjunctiva. Stock[2] produced a sclerosing keratitis and also a typical interstitial keratitis in guinea pigs. Desvaux[33] has called attention to white deposits low down in the cornea and deeply situated as characteristic of tuberculosis. It may be impossible to separate the tuberculous and leutic types; trauma seems to act as a predisposing cause in both. The Wassermann reaction, condition of parents, and stigmata of inherited syphilis, or signs of tuberculosis in other organs help to establish the diagnosis. As long ago as 1885 Roy and Alvarez[34] demonstrated giant cells and tubercle bacilli in the corneal parenchyma. Many subsequent reports prove beyond doubt that tuberculous keratitis occurs in the absence of any of the concomitant lesions of syphilis. Stock,[2] Verhoeff,[35] and Igersheimer[36] have produced it experimentally.

Scleritis is frequently due to tuberculosis and usually in young women. To what extent the old conception of rheumatic scleritis is justified is problematic. If "rheumatic" means due to some focus of infection, the term may still be useful; but there is considerable evidence brought forth by Verhoeff[37] for the tuberculous origin of scleritis.

He treated thirteen consecutive cases with tuberculin. All gave a general reaction; nine gave a focal reaction. Excised nodules from four cases showed epithelioid cells and occasional giant cells, but no batilli. Four cases improved steadily under tuberculin. It is proper to state that all the cases occurred in women and three showed exacerbations at the menstrual period. Interstitial involvement of the cornea was noted in all but two cases; this cleared more slowly than the scleral lesions and left traces. Three cases had a sluggish iritis.

In connection with the failure to find bacilli, Jackson's remark is pertinent, namely, that this failure scarcely raises any doubt regarding the diagnosis, since it is extremely difficult to find organisms in these chronic lesions. Indeed, so-called "surgical tuberculosis" rarely shows bacilli. Verhoeff's experiments with rabbits showed that tuberculous sclero-keratitis can be produced closely resembling the human disease. He believes that the aqueous is the source of infection through the filtration angle. Chou,[38] on the contrary, believed that in his case the sclera was involved directly from the blood vessels. Reference has been made to the successful inoculation of the rabbit's anterior chamber with tissue from scleral nodules (Collomb[7] and Lawford[6]). We should not, however, conclude that the evidence for a tuberculous etiology of scleritis negatives the possibility that other infections cause some cases. Benedict's[39] studies indicate that cervical infections cause some of the recurring cases.

The uveal tract is especially vulnerable to hematogenous infection. Rollet and Colrat[31] says that tuberculosis of this structure is like tuberculous meningitis; though clinically primary, it is always anatomically secondary. Stock's[2] rabbits always showed uveal involvement. Finnoff's[15] rabbits showed uveal lesions identical with those considered tuberculous in the human eye. No accurate figures are available as to its relative frequency. Germans find a great amount of uveal tuberculosis, even as high as 90 per cent of iritis cases; but their methods of study are inadequate and their tendency is to regard an intractable uveitis, which is not luetic, as tuberculous because of a general reaction to tuberculin without a focal reaction, which they avoid as unsafe. We should not be content without a much more elaborate search for focal infections than the Europeans make. Hessberg[40] states that tuberculosis has first place as the cause of diseases of the uvea, especially iritis which he attributes to tuberculosis in half the cases. Peters[41] cites eighty-eight cases of tuberculosis of the anterior segment of the eye, fifty-seven being definitely tuberculous and the others admitting of question. American statistics, notably those of Brown and Irons,[42] give a much smaller incidence of tuberculosis.

Nodular types of iritis are usually syphilitic or tuberculous. The peripheral location of nodules points to the latter cause. Hemorrhage is very frequent in tuberculous nodular iritis, according to Rollet.[31] The generalized iritis which accompanies tubercles of the sclera, pectinate ligament, and ciliary body is part of the toxic manifestation of tuberculosis.

The ciliary body is attacked by true tubercle, as Verhoeff[35] has shown, and he believes that many obscure cases (rheumatic) are really tuberculous; he obtained general and focal reaction to tuberculin.

Tuberculosis of the chorioid is well known. It may be miliary, due to meningitis, or it may be solitary.

De Schweinitz and Shumway[43] called attention to its occurrence in children as a more rapidly growing mass than the malignant tumor and with greater tendency to invade the sclera. Jessop[44] noted the scarcity of pigment and new blood vessels and the absence of vitreous opacity accompanying chorioidal tubercle. F. Lagrange[45] made the same observation and even stated that intra-ocular syphilis manufactures pigment whereas chorioidal tuberculosis destroys pigment. Rollet and Colrat[31] noted this in tuberculous iritis and show pictures of epithelioid cells acting as phagocytes taking up pigment from the iris. Lagrange also believed that visual acuity is less reduced by tuberculosis and scotomas are less frequent. Gilbert[46] regards tuberculosis as the dominant cause of chorioiditis dissemminata, which he believes to be hardly ever due to syphilis in adults.

It may be said that nodular lesions in the chorioid without great tendency to pigmentation or great clouding of the vitreous suggest tuberculosis. A later development of clear-cut, pearly, almost metallic-looking nodules is very suggestive. My colleague, Dr. John Dunn, called my attention to this, and Finnoff[15] found this appearance to correspond to the healing following the stage of caseation in chorioidal tubercles in the rabbit. Undoubtedly the early stages of suspected tubercles of the chorioid have been seen to resolve after focal infections have been removed; and this brings up several questions to be discussed later.

The course of certain insidious and progressive cases of tuberculous uveitis in which the second eye eventually is involved, together with the similarity of the histologic pictures, has suggested that sympathetic ophthalmia may be a tuberculous process.

Tuberculosis of the retina appears as minute perivascular lesions predisposed to the bifurcations of the smaller veins, often peripheral rather than central. At times a chain of tubercles lies along a vein. The tendency to hemorrhage is of course great; and a distinctly hemorrhagic disease has been associated with tuberculosis, namely the recurring retinal hemorrhages of adolescence. That other causes operate in this disease is shown in the literature, notably in Finnoff's[47] extensive review of the subject; but tuberculosis seems to be the main cause. It should also be noted that retinal detachment may occur over the area of a chorioidal tubercle, and even more widely, being quite extensive where conglomerate tubercle occurs in the chorioid. Embolism of the central retinal artery has been attributed to tuberculosis in at least one case (Oloff)[31] and thrombosis of the central vein a number of times.

The optic nerve head has been the site of tuberculosis according to several observers. A. Knapp[48] reported one case in 1903. Bartels[49] described tuberculous papillitis in five cases associated with neuro-retinitis reaching to the macula. He based the diagnosis on the great improvement following the use of tuberculin. Obviously the tuberculous etiology of retrobulbar neuritis is more difficult to establish (Chevallereau, Koulebyakine, Igersheimer[31]). Papillitis as a concomitant of tuberculous meningitis is mentioned by numerous authors. Gowers[31] believed it to exist in 25 per cent of cases and Heinzel and Parinaud[31] in 60 per cent of cases. Papillitis may also be the predecessor of the so-called pseudoglioma of the retina. The diagnosis of retinoblastoma (glioma of the retina) has led to the enucleation of eyes which proved to be the seat of massive tuberculous processes.

The Diagnosis of Ocular Tuberculosis.—The diagnosis of ocular tuberculosis involves more than the clinical appearance of certain lesions. It involves, first, a realization that such a thing is possible. Every obscure lesion, not explained otherwise, or not yielding to treatment in a reasonable time, should be regarded as potentially tuberculous. A general study with reference to tuberculosis elsewhere in the system should be instituted. It is perhaps not out of place to remind ourselves that intra-ocular inflammation, in the absence of trauma (and even in the presence of trauma oftentimes), is secondary to a source of infection elsewhere in the body. The types of ocular disease discussed above demand medical study. It must be understood, however, that the primary focus of tuberculosis need not be an extensive one: indeed ocular tuberculosis occurs in apparently healthy individuals. The chance lodgement of bacilli in an ocular capillary does not necessarily mean the breaking down of a vast number of tubercles. We deal with minute lesions in the eye, often individual tubercles; such lesions would have to be multiplied many fold before they would give the usual clinical evidence of tuberculosis in the chest, for example. It is sometimes difficult to make the medical man realize this; and one of our chief needs is to get the necessary co-operation in the study of such cases and the consequent treatment. My own belief is that a negative physical examination and history do not in the least invalidate the diagnosis, but merely exclude gross tuberculosis elsewhere than in the eye, and allow greater freedom in the use of tuberculin.

The Use of Tuberculin.—Of chief importance in the diagnosis of tuberculosis is tuberculin. We are all familiar with the great expectations aroused by Koch's first announcement of this agent for treating tuberculosis, and the unfortunate results of its injudicious use. There is still much argument as to the value and safety of the more refined methods of using tuberculin in such smaller doses than Koch first advocated. Koch's old tuberculin, known as O. T., is the most generally used preparation. The dosage varies according to the lesion in the eye, smaller doses being safer when the lesion is so located that a considerable reaction in the eye would be disastrous. This applies especially to the retina. For suspected tuberculosis of the anterior segment of the eye doses of 1 mg., 2, or even 5 mg., are given, with an interval of three to five days between doses. Some have recommended even a dose of 10 mg. Three types of reaction should be noted, general, local, and focal. The general reaction shows in fever and malaise such as one gets from the injection of various foreign proteins. A distinction should be made between the terms "local" and "focal;" this is not always done in the literature and confusion arises as to the writer's meaning. This can be avoided by using "local reaction" to mean the redness, swelling, and pain at the site of the subcutaneous injection, usually the arm. The "focal reaction" is the increase in activity of the suspected occu-

lar lesion. General and local reaction mean that the individual is sensitive to tuberculin and therefore has had a tuberculous focus at some time. A focal reaction indicates that the ocular lesion is tuberculous. I am aware that the specificity of the reaction is questioned, and it is not impossible that tuberculin is, after all, merely a form of foreign protein. The intradermal use of minute doses of tuberculin to determine the sensitivity of the patient is of value, especially in those cases where a considerable reaction in the eye would be harmful; the value of small doses, not producing focal reaction, in such cases may be very great. This aspect of tuberculin therapy will be considered in another paper in this symposium.* Dr. William H. Wilmer kindly permits me to refer to this method as used at the Johns Hopkins Hospital. The probability of an obscure ocular lesion's being tuberculous when the patient shows a high degree of sensitivity to tuberculin is so great that it justifies the diagnosis, and therefore the use of tuberculin therapeutically.

Treatment of Ocular Tuberculosis.—Two obvious rules apply in the treatment of ocular lesions regarded as tuberculous. First, the local treatment must be carried out which is indicated in inflammatory conditions of other etiology. Rest, atropin, dionin, heat and so forth, are as necessary here as in any other inflamed eye; indications and contra-indications will be the same. Second, the patient is tuberculous, and however slight (or absent) the signs of tuberculosis in other organs, the general care given to a case of tuberculosis is necessary. It is true that ocular tuberculosis appears in seemingly healthy individuals; if a severe tuberculosis exists the accompanying eye condition may be so serious as to demand enucleation, or may be disregarded by the medical attendants. The opposite condition, of good general health and moderately or extremely severe ocular disease, should not make us regard the case as exclusively in our province. The recent report of Derby and Carvill,[50] showing a mortality of 17 per cent in a series of 63 cases of ocular tuberculosis followed over a long period of time, indicates that this is far from a trivial matter.

Many methods of attacking the ocular condition have been advised. Guaiacol, iodoform, creosote, and mercurials have been injected into the anterior chamber and beneath the conjunctiva. Sterile air has had a vogue and been abandoned. Lundsgaard[51] gave phototherapy an extensive trial and was disappointed in it. Radiotherapy has been used with gratifying results by Rollet and Colrat.[31] They found it much more efficacious than tuberculin. Their careful and conservative reports deserve serious attention.

For lesions accessible to surgery, this is the method of choice. Chalazia, lid abscesses, lacrimal gland and sac lesions, and conjunctival growths can be excised. Likewise, extensive and incurable intra-ocular disease, which not only causes blindness but may itself act as a focus of infection endangering the system, demands enucleation of the eye.

The possibility of the association of tuberculosis and pyogenic infections in the same individual must be considered. We know that tuberculosis in general tends to heal, and the disastrous results of this disease come usually from secondary infections leading to the breaking down of healed tubercles and the dissemination of bacilli which were walled off. Therefore, the outcome of a case of ocular tuberculosis may be materially altered by attention to focal infections. Luedde,[52] while recognizing the value of tuberculin, believes that its use will be less and less as we attend more carefully to the radical elimination of foci of infection.

Tuberculin Therapy.—One could establish a convincing case for or against tuberculin by selecting his references from the literature. Most enthusiastic reports come from the German clinics; the French are, in general, much less enthusiastic. English and American observers are perhaps still less enthusiastic, with notable exceptions. It will probably be agreed that tuberculin if used therapeutically at all should be begun in minute doses and continued over a long period of time, with very gradual increase of dosage, avoiding any considerable reaction. The bacillen emulsion (B. E.) is the favorite preparation. Intervals between doses vary from three days to a week. Lesions of the posterior segment of the globe demand more care and smaller doses to avoid damage from increased activity which may mean hemorrhages and exudates destroying vision. By minute doses we mean 0.000,000,1 mg. of tuberculin, or 0.000,001 mg. A large dose in retinal tuberculosis for example, would be 0.000,1 mg. Much larger doses are reached in the prolonged treatment of tuberculosis of the anterior segment.

Jackson, Finnoff, Gamble, and Wilmer have obtained gratifying results with tuberculin and

*Dr. Alan Woods discussed this subject fully in his paper read on the same occasion, entitled "Tuberculin Therapy."

consider it a valuable agent, though they are not so enthusiastic as von Hippel and his followers. Verhoeff and Derby are skeptical of its value and lay chief stress on the general care of the patient. If I may be permitted a personal opinion based on eighteen years' experience with tuberculin, I would say that this is a valuable agent for increasing resistance and hastening the cure of ocular tuberculosis. Whether it be explained as a specific action or as the action of a foreign protein, its use is followed by a healing and quieting of the inflammatory state of eyes which we did not see improve in the days before tuberculin came into use. The observations made shortly after von Hippel's method of using tuberculin became known, when a large material was available at the Wills Eye Hospital, especially in the services of the late Drs. Risley and Ziegler, impressed me most forcibly. When the cases of massive tuberculosis accompanying well recognized, advanced disease elsewhere in the body, cases of tuberculous meningitis, and cases of external lesions amenable to surgery are omitted, there remains a large group of cases which are probably, and indeed almost certainly, tuberculous, as shown by diagnostic tests with tuberculin, cases perhaps of an attenuated infection without great virulence of the responsible organism, as Wilmer has emphasized, but sufficiently virulent to cause serious damage to the delicate structures of the eye. The outcome of such cases varies with the duration and extent of injury accomplished. Any means of increasing resistance, of hastening resolution of inflammatory material, and lessening destruction of normal tissues and scar formation, is valuable and may save vision which would be otherwise lost. I believe that tuberculin offers such a means, and that its judicious use is safe. It is of course possible to use it recklessly, and it is possible to neglect other measures. Both mistakes should be avoided.

CONCLUSIONS

Tuberculosis of the eye is sufficiently frequent to warrant most careful consideration and appropriate treatment. The intimate cooperation of the ophthalmologist and general physician, constantly demanded in the modern practice of medicine, is especially needed in treating this type of case. It is important to keep in mind the possible tuberculous etiology of all protracted ocular inflammations not explained on other grounds.

The relationship of focal infections to tuberculosis requires further study. To what extent do the former convert benign lesions into destructive lesions, and to what extent does the removal of infected foci help to cure tuberculous infection?

The use of the x-ray in the treatment of ocular tuberculosis deserves a more thorough trial than it has yet received.

The exact status of tuberculin, as to its specificity, or its function as a foreign protein, needs further investigation.

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Discussion follows paper of Dr. Woods, page 619.

THE THERAPEUTIC USE OF TUBERCULIN IN OCULAR TUBERCULOSIS*†

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The purpose of this report is to present our experiences with the therapeutic use of tuberculin in ocular tuberculosis. While such therapeutic utilization of tuberculin is of long standing and widespread use it seems timely to summarize the results obtained in different clinics. Especially is this true, since Derby and Carvill[1] have recently reported their experiences with tuberculin in anterior ocular tuberculosis, and have concluded that this treatment is practically without value. The discussion following the presentation of their carefully prepared report indicates that other observers do not altogether concur with their conclusion.

Prior to the first reorganization of the Eye Dispensary of the Johns Hopkins Hospital in 1923, suitable cases of ocular tuberculosis were treated with tuberculin in the Phipps Tuberculosis Clinic. The liaison between the two departments was subject to physical handicaps, and the records were consequently sketchy and incomplete. Since 1923 it has been possible to analyze accurately the results obtained in ocular tuberculosis with tuberculin therapy, and especially since the organization of the Protein Clinic at the Johns Hopkins Hospital under the direction of Dr. Leslie N. Gay and Dr. Nathan B. Herman, where cases of ocular tuberculosis have been treated. Our observations, therefore, do not extend over so long a period as those of Derby and Carvill. Yet, inasmuch as many cover a period of four years, and practically all have been carefully followed, an analysis of the results gives a fairly accurate indication of the value of tuberculin treatment in tuberculosis of the eye, and certainly indicates the initial response which may be expected.

*Read in Section on Eye, Ear, Nose and Throat, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

†From the Wilmer Ophthalmological Institute, Johns Hopkins Hospital.

HISTORICAL

The extensive literature has recently been abstracted by Clapp,[2] and Wilmer[3] has recently presented an excellent review of the types and pathology of eye tuberculosis. Notable reports on the results obtained with tuberculin are those of Igersheimer,[4] Derby,[5] von Hippel,[6] Weigelein,[7] Derby and Carvill[1] and Hartig.[8]

The experiences of these observers are shown in brief in *Table 1.* Four German observers report a total of 392 cases of various forms of ocular tuberculosis treated by tuberculin injections: 46.5 per cent are reported as healed, 37.3 per cent reported as improved, and 16.2 per cent, as unimproved.

TABLE I

Experiences of Other Authors with Tuberculin.

Author	No. of Cases	Healed	Improved	Unimproved
Igersheimer	34	32.3%	56.0%	11.7%
von Hippel	243	75.7%	17.3%	7.0%
Weigelin	77	41.6%	34.0%	24.4%
Hartig	38	26.3%	42.0%	31.7%
Total	392	46.5%	37.3%	16.2%

Hartig's report deserves especial attention. As a control of the 38 patients treated with tuberculin, he followed the course of 14 other patients with ocular tuberculosis who did not receive tuberculin. His observations were over one year. While 26.3 per cent of the tuberculin treated patients were healed, not one of the untreated patients was healed; only one patient (7 per cent) showed any improvement, while the remainder (93 per cent) were either without clear improvements or unimproved.

Hartig further reported two observations on the initial response to tuberculin therapy. Of 96 patients treated, 32.3 per cent showed clear

TABLE II. (Hartig)

Initial Response to Tuberculin Treatment.
Comparison of Cases of Ocular Tuberculosis Treated with Tuberculin, to Those not Treated.

	No. of Cases	Improved	No Clear Improvement	Unimproved
Tuberculin treated	96	32.3%	45.8%	21.9%
Tuberculin withheld	32	18.2%	59.0%	22.8%

improvement in one month, while of 32 untreated patients, only 18.2 per cent, showed any improvement. These results are shown in *Table II.*

SELECTION AND MANAGEMENT OF PATIENTS

After diagnosis of the ocular lesion, both private and dispensary patients were subjected to a medical survey, with especial attention to the presence of other foci of tuberculosis, and of non-tuberculous foci of infection, together with the usual laboratory examinations, including tuberculin tests. Non-tuberculous foci of infection, such as infected teeth, tonsils, sinuses, were at once treated. The patients in whom the eye lesion was clearly tuberculous were then placed on tuberculin treatment. Other patients, in whom definite non-tuberculous foci of infection were found, and in whom the etiology of the eye condition was not clear, were given the usual indicated local and general treatment, and kept under observation. When the subsequent developments indicated that the condition was probably of a tuberculous nature, the patients were then placed on tuberculin treatment. Cases in which a reasonable doubt existed are not included in this series. During the course of tuberculin treatment, the patients were kept under constant observation, the eyes were continually examined for any evidence of focal reaction, and the dose of tuberculin was regulated in accordance with any general or ocular reaction observed. The general hygienic conditions were improved as far as possible, and the medical needs of the patient met as indicated.

The tuberculin was continued either until healing of the lesions had occurred or until a complete course had been given, that is, the dose had been increased until the patient withstood an injection of about 60 mgs. without reaction. Many of these cases, especially those noted as "improved" remain under treatment.

A patient regarded as healed is periodically examined at intervals of about four months, and the tuberculin hypersensitivity tests repeated. Tuberculin therapy is resumed on the return of tuberculin hypersensitivity, even without any evidence of a recurrence of activity in the eyes. Thus a number of patients have had courses of tuberculin injections at intervals on a return of hypersensitivity, although their eyes showed no recurrence of the disease. It seems probable that this maintenance of a tuberculin immunity has a definitely beneficial effect in preventing the recurrence of tuberculosis in the eyes.

TUBERCULIN

Old tuberculin (O. T.) is used routinely in the diagnostic tests, and broth filtrate (B. F.) is used therapeutically. Both tuberculins are prepared and standardized in the Phipps Tuberculosis Clinic of the Johns Hopkins Hospital. These tuberculins are prepared as follows:

Preparation of O. T.—Tubercle bacilli are grown in a glycerinated broth for six weeks. The culture is then boiled for one hour. It is then filtered and passed through a Berkefeld filter. The filtrate, evaporated at 50°-54° C. over a water bath to one-tenth of its original volume, constitutes crude tuberculin, and is evaluated arbitrarily as 1,000 mg. per c. c. Dilutions are made on this basis. The tuberculin is tested by guinea pig inoculation for strength and viable bacilli.

Preparation of B. F.—Tubercle bacilli are grown in glycerinated broth for six weeks. Without heating, this is then filtered through paper and passed through a Berkefeld candle. The filtrate, B. F., is checked by guinea pig inoculation for strength and viable bacilli. This filtrate is also arbitrarily evaluated as 1,000 mg. per c. c., but as this solution is one-tenth as strong as the O. T. the solution must be labeled according to their strength, with the initials O. T. or B. F.

Stock solutions can be kept indefinitely without preservatives. When preserved with 0.25 per cent phenol, a precipitate of protein containing some of the active substance gradually forms, thus reducing the potency.

Tuberculin Tests.—(O. T.) The tuberculin test used in all cases was the intra-cutaneous tuberculin test. The initial dose was 0.1 c. c. of a 1:100,000 solution (0.001 mg.) If this was negative, 0.1 c. c. of a 1:10,000 solution (0.01 mg.) was used. If this was negative, 0.1 c. c. of a 1:1000 solution (0.1 mg.) was used. If this test was negative the patient was regarded as showing no abnormal or pathological hypersensitivity to tuberculin. In a few patients, strongly positive to 0.001 mg., a still weaker dilution, or 0.1 c. c. of a 1:1,000,000 solution (0.0001 mg.) was used.

Tuberculin Treatment.—(B. F.) For treatment, tuberculin solutions of different strength are prepared, called 9, 8, 7, 6, 5, 4, 3, 2, and 1. The strengths of these solutions are as follows: 9, 1 c. c.=0.00001 mg.; 8, 1 c. c.=0.0001 mg.; 7, 1 c. c.=0.001 mg.; 6, 1 c. c.=0.01 mg.; 5, 1 c. c.=0.1 mg.; 4, 1 c. c.=1.0 mg.; 3, 1 c. c. =10.0 mg.; 2, 1 c. c.=100.0 mg.; and 1, 1 c. c.

=1 mg. (straight tuberculin). The initial therapeutic dose is usually 0.1 c. c. of No. 8 (0.00001 mg.) In the extremely hypersensitive, the initial dose is 0.1 c. c. of 9 (0.00001 mg.). The dosage is then increased in the usual manner. The following protocol is the treatment of a typical case in which no reaction was noted, and where it was never necessary to reduce the dose:

PROTOCOL

Treatment of Mr. T. with tuberculin.—Injections given twice each week, subcutaneously in the deltoid muscle, with a tuberculin syringe.

Solution 9 (1/100,000 of a mg. per c.c.)
Dose 1—0.1 c.c.
2—0.2 c.c.
3—0.35 c.c.
4—0.5 c.c.
5—0.7 c.c.
6—0.9 c.c.

Solution 8 (1/10,000 of a mg. per c.c.)
Dose 7—0.1 c.c.
8—0.2 c.c.
9—0.35 c.c.
10—0.5 c.c.
11—0.7 c.c.
12—0.9 c.c.

Solution 7 (1/1000 of a mg. per c.c.)
Dose 13—0.1 c.c.
14—0.2 c.c.
15—0.35 c.c.
16—0.5 c.c.
17—0.7 c.c.
18—0.9 c.c.

Solution 6 (1/100 of a mg. per c.c)
Dose 19—0.1 c.c.
20—0.2 c.c.
21—0.35 c.c.
22—0.5 c.c.
23—0.7 c.c.
24—0.9 c.c.

Solution 5 (1/10 of a mg. per c.c.)
Dose 25—0.1 c.c.
26—0.2 c.c.
27—0.3 c.c.
28—0.4 c.c.
29—0.5 c.c.
30—0.6 c.c.
31—0.7 c.c.
32—0.8 c.c.
33—0.9 c.c.

Solution 4 (1 mg. per c.c.)
Dose 34—0.1 c.c. Begin treatments once each week.
35—0.2 c.c.
36—0.3 c.c.
37—0.4 c.c.
38—0.5 c.c.
39—0.6 c.c.
40—0.7 c.c.
41—0.8 c.c.
42—0.9 c.c.

Solution 3 (10 mg. per c.c.)
Dose 43—0.1 c.c.
44—0.2 c.c.
45—0.3 c.c.
46—0.4 c.c.
47—0.5 c.c.
48—0.6 c.c.
49—0.7 c.c.
50—0.8 c.c.
51—0.9 c.c.

Solution 2 (100 mg. per c.c.)
Dose 52—0.1 c.c.
53—0.2 c.c.
54—0.3 c.c.
55—0.4 c.c.
56—0.5 c.c.
57—0.6 c.c.
58—0.7 c.c.
59—0.8 c.c.
60—0.9 c.c.

When solution 4 is reached, the patient should receive treatment once a week.

When a general or local ocular reaction occurred during treatment, the dose was promptly decreased usually from four to ten levels, depending on the severity of the disease, and again increased as before with the patient continually under observation.

CLASSIFICATION OF CASES

The forty-two cases here reported are grouped under five general headings according to the predominating eye lesion: keratitis, kerato-iritis, scleritis, posterior ocular tuberculosis, (choroiditis, hemorrhagic retinitis, chorio-retinitis, so forth) and generalized uveitis. Phlyctenular keratitis is not included in this series, as children with this condition are usually treated in the Harriet Lane Home for Invalid Children, an integral part of the Johns Hopkins Hospital, and are seen by the staff of the eye department only in consultation. Inasmuch as children constitute the vast majority of these cases, such a report is not included in this survey.

The cases are classified either as unimproved, improved, or healed. Unimproved indicates that the disease either remained stationary or progressed; improved indicates that the process appeared to be definitely healing, although some activity was still present on last examination; healed indicates that the evidences of active inflammation subsided, leaving only evidences of an atrophic disease. These patients were all in this atrophic stage when last examined.

RESULTS

Keratitis.—The predominating lesion in the first group of patients here reported was keratitis. Several cases were complicated by more or less uveal disturbance, but the corneal lesions appeared to be salient. They comprise cases of parenchymatous keratitis, disciform keratitis, and bullous keratitis. The duration of treatment, complications, tuberculin tests, and results are shown in *Table III.* No patient was regarded as completely healed, inasmuch as corneal infiltrates still exist to a greater or less extent in every patient, although every one was sufficiently improved, to enable him to continue his usual routine of life without pain or undue photophobia. The vision in every case was improved and the inflammation had subsided in all except one

patient. Three patients are still under treatment.

TABLE III
Keratitis

Name	Age	Non-ocular T.B.	Other Comp.	Period of Observation	Intradermal Tuberculin Test	Duration of	Recurrences	Result
B.	49	0	*	8 mos.	1/100 mg.+	1 course	1	Imp.
H. 39336	16	0	0	18 mos.	1/100 mg.+	1 course	0	Imp.
H. 47045	36	0	0	10 mos.	1/100 mg.+	1 course	1†	Imp.
H. 66486	30	0	0	3 mos.	1/1000mg.+	1 course	0	Imp.
H. 74501	26	0	0	4 mos.	1/100 mg.+	1 course	0	Imp.

*Infected tooth (extracted).
†During treatment

Scleritis.—The two patients with early typical nodular scleritis showed no other tuberculous or non-tuberculous foci of disease. They showed prompt healing under tuberculin without recurrence.

Iritis, Kerato-Iritis.—Of the four patients in this group, one with nodules of miliary tuberculosis on the pupillary edge of the iris, has been under treatment for one year and appears healed, without any activity. The remaining three are still under treatment. Two are definitely improved and one is unimproved.

TABLE IV
Scleritis

Name	Age	Non-ocular T.B.	Other Comp.	Period of Observation	Intradermal Tuberculin Test	Duration of Treatment	Recurrences	Result
S. S.	35	0	0	14 mos.	1/1000mg.+	1 course	0	*
H. 25224	56	0	0	2½ yrs.	1/1000mg.+	1 course	0	*

*Healed

Uveitis.—Eight patients in the series showed a general uveitis, unilateral in 6, bilateral in 2 instances. Two patients, who have had prolonged treatment over two years or more appear healed. Two patients, all under treatment a much shorter time, are greatly improved; and only one, under treatment for one year, is unimproved. One patient suffered a relapse under treatment, apparently a focal reaction after tuberculin administration, and one had a recurrence after early improvement.

TABLE V
Iritis and Kerato-Iritis

Name	Age	Non-ocular Tuberculosis	Other Complications	Period of Observation	Intradermal Tuberculin Test	Duration of Treatment	Recurrences	Result
A. K.	61	0	†	1 year	1/10000mg.+	1 course	1*	Healed
H. 64623	39	0	0	4 mos.	1/1000 mg.+	1 course	0	Unimproved
H. 58294	31	0	0	4 mos.	1/100 mg.+	1 course	0	Improved
H. 46692	41	0	0	5 mos.	1/100 mg.+	1 course	0	Improved

*During treatment.
†Infected tonsils removed.

TABLE VI
Uveitis

Name	Age	Non-ocular Tuberculosis	Other Complications	Period of Observation	Intradermal Tuberculin Test	Duration of Treatment	Recurrences	Results
M. McC.	28	0	0	2½ yrs.	1/1000mg.+	1 course	0	Healed
R. P.	37	0	0	1 year	1/100 mg.+	1 course	0	Unimproved
E. P.	27	0	*	6 mos.	1/1000mg.+	1 course	0	Improved
C. R.	37	0	**	4 mos.	1/1000mg.+	1 course	1	Improved
H. 57571	52	0	0	1 year	1/100 mg.+	1 course	0	Improved
H. 73793	37	0	***	4 mos.	Positive	1 course	0	Improved
H. 62807	32	0	**	8 mos.	1/1000mg.+	1 course	1†	Improved
H. 19273	36	0	0	2½ yrs.	1/1000mg.+	1 course	0	Healed

*Pelvic infection (Operated)
**Infected tooth (Extracted)
***Gingivitis
†During treatment

Posterior Ocular Tuberculosis.—In this group of 23, are included exudative tuberculous choroiditis, chorio-retinitis, with the formation of actual tubercles, and the hemorrhagic retinitis of young adolescents. The highest percentage of good results was observed in this series: 61 per cent apparently were definitely healed with no evidences of activity, and 30 per cent were definitely improved. Seven patients in this group showed definite recurrences, two of which showed later complete healing after further tuberculin therapy. The remaining two patients were greatly improved, but cannot yet be regarded as healed. Only two patients in this group, 8.7 per cent, were unimproved. It is noticeable that only in patients with posterior ocular tuberculosis did we find other systemic evidences of tuberculosis. Four patients of this group, or 18.2 per cent, showed tuberculosis elsewhere in the body. The greater relationship with systemic tuberculosis may be explained on the premises of vascular metastasis, yet it is peculiar that in the eight patients with uveitis, no evidence of systemic tuberculosis was found.

The Series as a Whole.—In the entire series are forty-two patients. Four patients, or about 10 per cent, showed systemic evidences of tuberculosis. Sixteen patients, or almost 40 per cent, showed other foci of infection, usually infected tonsils, sinusitis or apically infected teeth, which might well be the predisposing causes to the outbreak of the local ocular tuberculosis. Ten patients, or about 25 per cent, had definite recurrences of the ocular disease after an initial healing or improvement. Nineteen patients of the series, or 45 per cent, appear healed. Nineteen, 45 per cent show a definite improvement, and may reasonably be expected to progress to healing. Four patients, or about 10 per cent,

TABLE VII

Posterior Ocular Tuberculosis (Choroiditis, Hemorrhagic Retinitis)

Name	Age	Non-ocular Tuberculosis	Other Complications	Period of Observation	Intradermal Tuberculin Test	Duration of Treatment	Recurrences	Result
T.	22	0	0	7 mos.	1/1000mg.+	1 course	0	Healed
L.	45	0	Infected teeth (Extracted)	19 mos.	1/1000mg.+	1 course	0	Improved
R.	42	0	0	2½ yrs.	1/1000mg.+	1 course	0	Healed
M.	32	0	Morphia addict	4 yrs.	1/1000mg.+	1 course	0	Healed
H.	61	0	Infected teeth (Extracted)	2 yrs.	1/10000mg.+	1 course	0	Healed
H.	30	0	Tonsils infected (Removed)	4 yrs.	1/10000mg.+	2 courses	1	Healed
C.	23	0	0	4 yrs.	1/1000mg.+	1 course	0	Healed
C.	38	0	Return of Tuberculin Hypersensitivity	2½ yrs.	1/1000mg.+	2 courses	0	Healed
C.	56	0	Infected Tonsils (Removed)	3 yrs.	1/1000mg.+	2 courses	1	Healed
C.	25	Healed apical lesion Family History)	Infected Tonsils (Removed)	3 yrs.	1/10000mg.+	2 courses	1	Healed
H. 72479	40	?	0	4 mos.	1/100mg.+	1 course	0	Unimproved
H. 60507	19	0	0	11 mos.	1/100mg.+	1 course	0	Healed
H. 40124	32	0	Infected Tonsils	4 mos.	1/100mg.+	1 course	0	Improved
H. 63525	32	0	Infected Tonsils (Removed)	9 mos.	1/100mg.+	1 course	0	Improved
H. 64800	27	0	Pyorrhea	8 mos.	1/100mg.+	1 course	0	Unimproved
H. 63572	27	0	Sinusitis	7 mos.	1/100mg.+	1 course	2 During Treatment	Improved
H. 29026	19	0	0	3 yrs.	1/1000mg.+	2 courses	1	Healed
H. 31311	17	0	0	1½ yrs.	1/100mg.+	1 course	0	Improved
H. 40124	32	Healed rt. Apex	0	20 mos.	1/100mg.+	1 course	0	Improved
H. 71925	26	Adenitis	Infected Tonsils (Removed, Sinusitis (Operated)	4 mos.	1/100mg.+	1 course	0	Improved
H. 51643	48	0	0	9 mos.	1/100mg.+	1 course	0	Healed
H. 63499	28	0	0	9 mos.	1/100mg.+	3 courses	1	Healed
C.	53	0	Sinusitis	3 yrs.	1/100mg.+	3 courses	1	Healed

TABLE VIII

The Series as a Whole

Disease	No. of Cases	No. Complicated By Other Tuberculosis	No. Complicated By Other Focal Infections	Recurrences	Result		
					Healed	Improved	Unimproved
Posterior ocular Tuberculosis	23	4 18.2%	10 45.4%	7 30.0%	14 60.9%	7 30.3%	2 8.7%
Uveitis	8	0	4 50.0%	2 25.0%	2 25.0%	5 62.5%	1 12.5%
Iritis	4	0	1 25.0%	1 25.0%	1 25.0%	2 50.0%	1 25.0%
Scleritis	2	0	0	0	2	0	0
Keratitis	5	0	1 20.0%	2 40.0%	0	5 100.0%	0
Total	42	4 9.5%	16 38.0%	10 24.3%	19 45.3%	19 45.3%	4 9.5%

show no improvement. *Table VIII* shows these results in tabular form.

In interpreting the results of tuberculin therapy in ocular tuberculosis, the time element is of especial interest. Seven of the forty-two cases reported have been under observation and treatment for three years or more. Of these seven patients, six appear definitely healed, while the remaining patient, with tuberculous choroiditis, although greatly improved, still shows some evidence of activity. Six patients have been under observation for from two to three years, and all of these patients appear healed. Eight patients have been observed for a period of from one to two years. Three of these patients can be classified as healed, four are improved, and one is unimproved. Twenty-one patients have been under observation and treatment for a period of less than one year. Only four of these patients appear healed, fourteen show definite improvement, while three are definitely unimproved. These statistics appear to indicate that while a rather prompt improvement in the clinical picture may be expected after the inauguration of tuberculin therapy, yet actual healing requires a rather long period of time. Especially does this appear true in corneal and posterior ocular tuberculosis.

TABLE IX

Time Element in Relation to Therapeutic Result

Periods of Observation	No. of Cases	Healed	Improved	Unimproved	Recurrences
3 years or more	7	6	1	0	5
2-3 years or more	6	6	0	0	0
1-2 years	8	3	4	1	1
Less than 1 year	21	4	14	3	4

Recurrences.—Ten patients had recurrences of the ocular tuberculosis, two late in their disease, associated with a return of the tuberculin hypersensitivity, and eight early in their disease. Of the recurrences in patients observed over five years, three occurred in the early period of observation. In the patients observed over one year only four showed recurrences. These figures indicate that recurrences are more likely to occur early in the disease, after initial improvement, and are somewhat at variance with those reported by other observers. These observations are too few, however, to permit conclusions to be drawn.

Mortality.—None of the patients of this series died.

Methods of Treatment.—Our observations on these patients, and others previously treated, indicate that the most important point in the specific treatment of ocular tuberculosis is that the early injections of tuberculin be minute. Several cases of ocular tuberculosis have been observed where the focal reaction occurring after even moderately small doses has been of such violence that the eye was damaged. Especially does this apply to the diagnostic use of tuberculin. While in other forms of tuberculous infection, such as osteomyelitis, diagnostic injections of 0.1 or 0.5 mgs. may be of value in suspected ocular tuberculosis, such injections seem dangerous and unwarranted. Even though of great diagnostic value, focal reactions in the eye from tuberculin should be avoided. During the therapeutic stage, it is important that the eye be kept under constant observation. Impending focal reactions are usually heralded by slight increases in the inflammation after smaller doses than would evoke a definite focal reaction of any intensity. Thus by constant observation the dose of tuberculin can be safely increased if the ocular inflammation is regressive, or it can be held at the same level or decreased if there is any increase in the inflamma-

tion from 24 to 48 hours after the administration of tuberculin.

Return of Hypersensitivity.—After a course of tuberculin therapy has been satisfactorily completed and the local ocular condition appears healed, there is frequently a return in the patient's skin hypersensitivity after the cessation of tuberculin injections. This return may be either rapid, in several weeks, or very slow, over a period of months or years. In several patients who have shown ocular recurrences, the return of the hypersensitivity has been very rapid, and associated with an outbreak of the ocular tuberculosis. It seems reasonable to assume that in these patients the ocular tuberculosis was quiescent and not healed. We have made clinical use of this observation. Patients who have completed a course of tuberculin injections with apparent healing of the eye lesions, are kept under observation and at intervals of four months, when possible, are again tested for skin hypersensitivity. Upon the recurrence of marked skin hypersensitivity, even though the eye is entirely quiet, in several patients we have again given tuberculin injections. In the five patients thus treated, we have observed no recurrences. It is possible that by this method the frequency of recurrences may be greatly lessened. In any event, even after healing of the ocular lesion has occurred, tuberculin injections should be continued for several months to prevent early recurrences.

Therapeutic Action of Tuberculin.—The manner in which tuberculin acts as a therapeutic agent is not definitely known, whether as a specific desensitizing and immunizing antigen, or by augmenting of the specific local and general resistance by a succession of small stimuli, is an open question. It appears to us that the latter explanation is the most plausible. That it is non-specific protein therapy does not appear a tenable hypothesis. It is true that a focal reaction may be produced in a non-tuberculous lesion after massive doses of tuberculin, and here the reaction is doubtless non-specific. But here tuberculin acts as would any protein. Focal reactions are not observed in non-tuberculous lesions by minute or even moderate doses of tuberculin, while such small doses frequently produce focal reactions in actual tuberculous lesions.

CONCLUSIONS

Our observations on tuberculin therapy in ocular tuberculosis have convinced us of its value. A number of patients reported in this series had pursued a steady down-hill course until tuberculin therapy was begun. It is true that without tuberculin, while the attacks of the disease are somewhat self-limited, recurrences are constant rather than unusual, and the clinical prognosis is usually definitely bad. Under tuberculin therapy a more rapid limitation of the attack may be expected, recurrences becoming rare rather than the rule, and apparent complete healing is observed in a large number of the patients.

SUMMARY

Forty-two patients with ocular tuberculosis have been treated with tuberculin. All patients were markedly hypersensitive to tuberculin given intracutaneously. Forty-five per cent of the patients, thus treated appear healed; 45 per cent show definite improvement; and 10 per cent are unimproved. The healing is slow, and the greater number of patients classed as "healed" are those treated over several years. About 25 per cent of the patients treated, showed recurrence of the local disease. Improvement usually begins early in the tuberculin treatment. These observations indicate tuberculin therapy to be of real value in the treatment of ocular tuberculosis.

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DISCUSSION (*Abstract*)

Papers of Dr. Hill and Dr. Woods.

Dr. F. Phinizy Calhoun, Atlanta, Ga.—My experience with this condition has been none too large and rarely have I had the opportunity long to observe patients affected with ocular tuberculosis. In private practice the disease is seldom seen, while in my clinic work (composed largely of negroes) it is by no means so common as one might imagine, and the follow-up of these patients as you know is difficult. I have observed ocular tuberculosis more frequently in the mulatto than in the pure black, and the structures most commonly involved are the cornea and sclera.

The frequently appearing publications on tuberculosis of the eye show the interest that is being taken in the subject. This is particularly true of the German school, where the idea of the prevalence of tuberculous eye lesions is rapidly gaining ground among ophthalmologists. It may be difficult for them to get away from their old adage that every one sometimes in his life has tuberculosis. And yet, on the other hand, are we not too enthusiastic in our cry about focal infections?

Bergmeister in his recent book, expresses the belief that the regionary lymph nodes may remain depots of virulent bacteria for many years until the accidental break into the circulation. Rollet and Colrat are in accord with this view for they believe that ocular tuberculosis is only a manifestation of a deep infection which is habitually latent.

The idea is substantiated by Finnoff, who was able in his experimental work to produce in rabbits practically all the types of ocular tuberculosis that occur in the human eye. He concluded that intra-ocular tuberculosis in the majority of cases is hematogenous in origin and must be secondary to a tuberculous focus elsewhere in the body. Further we are told that tuberculosis of the uvea is secondary and the primary focus is situated in the peri-bronchial lymph nodes evidenced by definite x-ray shadows at the base of a lobe. If this diagnosis is made solely from x-ray examination of the chest, I am skeptical of it, for we know that definite shadows are frequently found in the chests of apparently healthy individuals.

Tuberculin used routinely for diagnostic purposes without the usual preliminary physical examination, and therapeutically without definite reason, I believe is dangerous. I recall an instance in our city, where a severe peritonitis developed after an initial dose of tuberculin.

It is well to divide ocular tuberculosis into that which affects the anterior segment (the cornea, sclera, iris and ciliary body) and the posterior segment (the chorioid, the retina and its vessels, and the nerve). My impression is that infections of the anterior segment are more frequent and the diagnosis more readily made. Furthermore I believe that one may proceed with reasonable safety in the diagnostic and therapeutic use of tuberculin in such infections and that the clinical results are more satisfactory.

Intractable cases of irido-cyclitis, in which an occasional flare-up in the sclera can be explained as a metastasis from the uveal infection into the anterior perforating vessels, I regard as tuberculosis, assuming of course that other causes are ruled out. I have been able to substantiate this statement in the examination of an eye that came to enucleation. And here I wish to endorse two statements made by Dr. Hill:

(1) "Every obscure eye lesion not yielding to treatment in a reasonable time, should be regarded as potentially tuberculous."

(2) "Negative physical examinations do not invalidate the diagnosis of tuberculosis, but merely exclude gross tuberculosis and allow greater freedom in the use of tuberculin."

For lesions involving the posterior segment, especially those of the macular region and retinal vessels, the diagnostic doses of tuberculin are contra-indicated, for the reason that fresh exudates and hemorrhages into the retina are known to occur with disastrous results. Therapeutically, in such cases tuberculin should be administered with the greatest caution and not until syphilis and every possible focus has been eliminated. Of importance is the frequent ophthalmoscopic inspection of the fundus. I agree with those who have written extensively, that our standard text books on ophthalmology have not devoted enough space for a description of tuberculosis of the retina and the vessels. Those who wish to be informed must consult the literature. I have seen in a stout healthy female, with what was clinically a solitary tubercule of the iris, a sharp local, focal and general reaction to 0.02 mg. of Koch's old tuberculin. The lesion disappeared in two days. Would not any non-specific protein have produced the same reaction? Curiously, there was a recurrence of the iritis with a similar reaction to 0.01 mg. old tuberculin, six years later. The specificity of sera, and tuberculous allergy, are questions too broad for me to introduce, and frankly they are to me hardly more than medical terms.

It is a common observation that in many instances of ocular tuberculosis, the removal of a focus of infection may lead to rapid improvement. The system has been flooded with so-called toxins which interfere with Nature's process of healing. When the scource is eliminated the rapidity of cure is at times startling. Only when the cause is obscure do I employ tests to determine the presence of tuberculosis.

The more I follow those who have had the opportunity to study seriously and follow their cases of ocular tuberculosis over a period of years, as Maude Carvill has shown in her admirable work, the more am I convinced that a certain number of cases do best under sanatorium care, in addition to the local eye treatment. The idea is rather an extreme one, but as additional reports are announced, I believe it will be more generally accepted.

I agree with Luedde who drew the following conclusions:

(1) A focal ocular reaction caused by a test injection of tuberculin renders the diagnosis of ocular tuberculosis highly probable, but not absolutely positive.

(2) The therapeutic benefit obtained from the use of tuberculin in ocular tuberculosis can be explained rationally as a specific or non-specific effect.

(3) The radical elimination of focal infections especially those of the naso-pharynx, and the proper treatment of any co-existing constitutional disease will render less and less frequent the indication for the use of tuberculin in ophthalmic practice, either as a diagnostic or a therapeutic agent.

Dr. Robert J. Warner, Nashville, Tenn.—The conjunctival test of Calmette should not be used in cases of suspected eye disease. The intracutaneous method of administering the tuberculin test is the one of choice, and should be carried out only in a hospital where the patient is kept for two days before the test, so that a two-hour temperature chart may be kept. During these two days there should be not more than one degree variation in temperature. I have had no case in which the old tuberculin diagnostic dose did any harm.

A diagnosis of ocular tuberculosis in the living eye should not be made without a positive tuberculin test, and a negative intracutaneous test properly performed and controlled excludes tuberculosis except in rare instances.

My experience agrees with that of Dr. Woods, that posterior ocular tuberculosis is the most prevalent form and of that form the hemorrhagic retinitis of adoles-

cence is most frequent. It is also the form that responds best to tuberculin treatment. The solitary tubercule of the chorioid offers the worse prognosis. Most of these eyes are lost and life is endangered though the presence of tuberculosis elsewhere in the body.

X-ray, radium, cautery and such treatments should be used along with the tuberculin.

Dr. Woods' percentages of healed, improved and unimproved cases agree with those of other authorities upon the subject.

Dr. Shaler Richardson, Jacksonville, Fla.—Liaison is necessary between the departments or the various specialists handling tuberculous patients. Every suspected tuberculous patient should be surveyed with meticulous care, by specialists. Indiscriminate tuberculin tests and the use of tuberculin therapy before a thorough physical examination has been made will occasion far greater harm than good. The value of intradermal tuberculin tests has been the subject of much controversy. Dr. John E. Weeks in a paper read before the Colorado Congress of Ophthalmology in 1925 said that the intradermal tuberculin test was of no value in the diagnosis of ocular tuberculosis. He relies upon the subcutaneous tuberculin test and considers it positive in diagnosing ocular tuberculosis only when a focal reaction is obtained. It has been my experience that the intradermal test is a highly sensitive one and of great diagnostic aid. Focal reaction of course is much more conclusive but is not essential when the eye condition is clinically tuberculous with a positive intradermal test.

Derby and Carvill's report of the result of tuberculin treatment in a group of cases of anterior ocular tuberculosis followed over a period of years certainly does not coincide with the results of other observers, and my experience in the treatment of such conditions would lead me to believe that tuberculin is of inestimable value in the greater number of cases.

Dr. Woods' scale of dosage is a workable one. I have under observation at the present time two patients with a tuberculous uveitis, both of whom are responding to tuberculin therapy. I am using the B. F. prepared by the Protein Clinic at Johns Hopkins and the dosage scale suggested by Dr. Woods. Let us not forget the value of the temperature curve in the diagnosis of ocular tuberculosis. In doubtful cases a carefully recorded temperature chart will in many instances aid us greatly in making a diagnosis.

Aside from the use of tuberculin in ocular tuberculosis we should not overlook the necessity of general hygienic care. Derby and Carvill in the analysis of their cases of anterior tuberculosis conclude with the following remarks: "And especially we urge the view that these patients should be regarded as seriously affected with tuberculosis and treated not only to cure the present attack, but to avoid recurrences and so to build up their resistance that they will have a reasonable certainty of avoiding a more serious form of tuberculosis later on."

Dr. J. W. Jervey, Greenville, S. C.—Not very long ago I had the pleasure of listening to a distinguished ocular pathologist who stated that it was impossible to make a clinical diagnosis between sympathetic uveitis and tuberculous uveitis, and intimated that in any event a clinical diagnosis of sympathetic uveitis was impossible. Shortly thereafter he arose on the floor of the same organization, at the same meeting, and reported eight or ten cases of sympathetic uveitis which he had cured by massive doses of diphtheritic antitoxin. I think we are in much the same position when we speak of the therapy of tuberculous uveitis. One suspects that some of the cases reported as being treated with tuberculin were not proved to be tuberculosis. We find ourselves reduced in many instances to the point of making a diagnosis by exclusion, and we revert to methods of a previous generation and try our hand, as they did in cases of suspected syphilis, and apply the therapeutic test. I believe in the therapeutic value of tuberculin. I have seen many cases of tuberculous condition of the eyes that have distinctly and decidedly improved, and one case in particular which comes to mind was one in which a very distinguished oculist did a complete exenteration of the orbit for supposed malignancy in a case which subsequently developed identically the same condition in the other eye. Since the eye had not responded to any other form of treatment, I tried tuberculin, and the eye, with a certain amount of vision, was saved.

Dr. J. A. Stucky, Lexington, Ky.—In my clinical work in trachoma in the mountains of Kentucky it was suggested by Dr. Anna Von Sholly of New York City, who was with me and later by Dr. Harlan, of Baltimore, that many of the cases of phlyctenular keratitis that did not improve after grattage might be tuberculous. I am inclined to agree with them. Later, by improving the condition of nutrition of these patients, giving them cod liver oil and a balanced diet the eye conditions were much improved and the trachoma and keratitis cured. I believe the point that glioma is possibly an intra-ocular tuberculosis is a good one, and that glioma is an intra-ocular evidence of deficient nutrition, just as trachoma is extra-ocular evidence. I have obtained better results with non-specific protein therapy and the use of cod liver oil than with anything else. If a positive diagnosis of glioma is made, radical surgery is indicated.

Dr. Edward Jackson, Denver, Colo.—One point in the therapeutics of ocular tuberculosis has not been mentioned. Dr. Hill referred to the experience of Lundsgaard as to the failure of heliotherapy and phototherapy. That failure was the failure of local heliotherapy and not what has been applied in the last few years: light baths and ultra-violet rays. In the last year in the *Transactions of the Ophthalmic Society of the United Kingdom* there have been two striking papers upon this subject, one by Mr. Charles Goulden of London, and the other by Duke Elder. Their results run parallel with those obtained at the Fitzsimmons Hospital in Denver. The therapy employed by Gould and Duke Elder was the light "baths," about one-fourth of the body being exposed, not to the sun, but to lamps that are more rich in ultra-violet radiation than is sunlight.

In phylctenular disease of the eye, and in tuberculosis chiefly of the anterior segment, the results were strikingly good in the cases in their series.

Dr. F. C. Thomas, Lexington. Ky.—I wish to emphasize the suggestion of Dr. Woods that the dosage of tuberculin be kept low. I have had the privilege for the last eight months of observing one of Dr. Wood's patients while she was under the course of treatment outlined by him. She started with a very infinitesimal dose, and at one time she had frank chorioidal exudate in both eyes. Vision in the right eye was 20/60; left, 20/40. She has had a troublesome general reaction even from these small doses and the dosage has had to be dropped back on several occasions. At present the vision in the right eye is 20/30 with the same glasses; and in the left, 20/20, and all that remains in the

fundus of either eye is several small spots of pigment in the upper temporal quadrant. There is no further evidence of exudate.

Another case several years ago had a patch of exudate that covered the macula. The vision was reduced to 20/200. She was given 0.0001 mg. tuberculin, following which she had a violent general reaction and was in bed several days. We have not given her the second dose, principally on account of the reaction, but the exudate has diminished at least to the extent that her vision in one eye is 20/30. The other eye is blind and has been for twenty years. This woman is carrying on now with very useful vision.

Dr. W. D. Gill, San Antonio, Texas.—The fact that we get improvement in uveitis following the administration of tuberculin does not necessarily mean that the uveitis is tuberculous. Non-tuberculous uveitis may respond favorably to tuberculin therapy and this is apparently due to the fact that tuberculin provokes a non-specific antibody response. The so-called therapeutic test is not an infallible diagnostic criterion.

We should not overlook the fact that a patient with extra-ocular tuberculosis may have a uveal inflammation secondary to a hidden pyogenic focus elsewhere in the body which will demand appropriate measures for its eradication.

Dr. Jackson's statement concerning the value of actinotherapy in the treatment of uveitis in general is certainly most important. Actinotherapy used intelligently, whips up the patient's defensive mechanism, produces a leucocytic response, and in all probability there is an associated increase in anti-body formation. Overuse is as bad, if not worse, than failure to use actinotherapy, as it may cause the patient to enter a negative phase in which the defensive mechanism of the body is decreased rather than increased.

Dr. Woods (closing).—The two chief points which have been brought out in the discussion are first, the non-specific reaction of tuberculin, and second, the use of chemophototherapy. As concerns the possible non-specific reaction of tuberculin, I do not believe this is the tenable hypothesis. Non-specific protein reaction is, of course, characterized by a febrile reaction and a general reaction, the mobilization of the leucocytes, and the occurrence of a focal reaction with other phenomena which have been well worked out. I think it is true we do not know which specific factors are responsible for the therapeutic results obtained in the non-protein reaction, yet, in general, we are quite safe in saying it is a stimulation of the organism by actual shock. This shock reaction is only obtained by giving a definite mass of protein and the actual amounts of protein given, even with the mildest application of the non-specific protein reaction, are a millionfold and more, greater than the amount of protein given in the minute doses which one employs with tuberculin. A non-specific protein, such as milk or diphtheria anti-toxin given in the same relative doses that tuberculin is given, would produce no reaction in the normal patient. I think probably we must attribute the definite reactions which are observed after minute quantities of tuberculin not to a non-specific reaction, but to a definite specific reaction.

Dr. Jackson has spoken of the use of chemophototherapy. We have recently used ultra-violet light in cases of anterior ocular tuberculosis with very good effect. Our experiences so far are not sufficient to present any detailed report on this subject, but in general the application of chemophototherapy has been followed by general improvement in the pathological picture in the anterior structures of the eye.

The point to be noted in the care of ocular tuberculosis is the management of the general hygiene of the patient. In the series just reported their medical needs were met as indicated, foci of infection were eradicated as far as possible, dietary errors were corrected and the general hygiene improved as much as could be done. Even when these factors are considered it appears to us that cases treated with tuberculin do much better than untreated cases and we are convinced that tuberculin has a decided beneficial effect in ocular tuberculosis.

TREATMENT OF PERFORATIVE APPENDICITIS WITH OR WITHOUT ABSCESS*

By Byrd Charles Willis, M.D., F.A.C.S.,
Rocky Mount, N. C.

To treat appendicitis ideally one should begin with the first symptoms, but this is rarely possible since the laity has not been educated by the profession concerning these. The diseased appendix never perforates without warning. The symptoms may be slight or fulminating, lasting for a few hours or for several days. In one case of the series reported here the operation was performed three and a half hours after the onset of pain. Perforation had occurred and there was localized peritonitis.

If the appendix perforates within twelve hours the pain is so severe that the patient is usually forced to seek medical aid, and this often affords the physician a golden opportunity to save life by means of competent advice with regard to surgical measures. There is a definite increase in the mortality rate in fulminating cases in which there has been improper treatment, according to the length of time elapsing between the appearance of symptoms and the operation, as in cases of intestinal obstruction, although not in the same ratio. Many patients with perforative appendicitis have previously had one or more mild attacks and recovery emboldens them to disregard medical advice. Those patients are likely to come to operation late and after purgation. Many other patients believe their pain is due to a certain food, and they purge themselves early without consulting their physicians, and before protecting adhesions have formed which render the appendix immobile. Of the twenty-three patients in this series who

*Read in Section on Surgery, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

died, sixteen had received from one to many purgatives. In my opinion, there is no more potent cause for increased mortality than purgation. It is greatly to the credit of the medical profession that it is disregarding the earlier teaching of operation as a last resort, and also that the dangerous and pernicious habit of purging has fallen into disrepute.

Surgeons agree almost unanimously that in acute cases of appendicitis operation should be performed in the first thirty-six hours, provided the patient is otherwise healthy, but after this period followers of the renowned Ochsner believe in tiding over certain types of cases. In some of these an abscess develops, in others there is improvement, and in a small percentage of cases the patients die. Many surgeons operate as early as possible after a diagnosis has been made, regardless of the time.

A questionnaire was addressed to all the members of the Southern Surgical Association as follows:

"Do you operate in all acute cases immediately after diagnosis is made regardless of the time elapsing since the attack began? Do you remove the appendix in all cases?" Their replies to each question are classified under yes, no, and yes with exceptions. To the first question, fifty-nine replied yes, twenty-seven no, and sixteen yes with exceptions. To the second question, seventeen replied yes, seventy-three no, and fourteen yes with exceptions. Two physicians failed to reply to the first question. Many answers were guarded, and to analyze them required careful study.

Unquestionably the majority of the surgeons who answered the questionnaire operate immediately in all acute cases. Many do not remove the appendix if there is a complicating abscess, believing that it is better to leave the appendix if it does not readily come to hand. This practice has been followed by Dr. Boice and me except in rare cases in which there are other complicating conditions that make a local or general anesthetic hazardous.

From July, 1914, to August, 1927, operation has been performed in 382 cases of acute perforative appendicitis at Park View Hospital; twenty-three patients (6 per cent) died. In all of these cases drainage was carried out. In a few cases the appendix was perforated and securely walled off by omentum; this was not detached, but was amputated with the appendix. There was no drainage in these cases. They are not included in this series. Peritonitis is not discussed because of the probability that in all cases of perforative appendicitis there is more or less peritonitis. The extension of the peritonitis has been the source of the greatest mortality in this series.

During this same period 1730 patients were operated upon, primarily for appendicitis, with thirty-two deaths, a mortality of 1.8 per cent. Six hundred twenty-four appendectomies were secondary.

Seventy-eight of the 382 patients were aged fourteen years or less; five (6.5 per cent) of these died. Eighteen (6 per cent) of the 304 adults died. In 299 of the 382 cases the appendix was removed primarily; fourteen of the patients (4.7 per cent) died. In eighty-three cases it was thought best simply to institute drainage, owing to the difficulty of removing the appendix or to the desperate condition of the patients. Nine of these (11 per cent) died. Of the 294 white patients, fourteen (4.8 per cent) died, and of the eighty-eight colored patients, nine (10 per cent) died. This mortality in the colored may have been due to late hospitalization; many of the patients were in a desperate condition, with spreading peritonitis following violent purging. The patients were all operated upon by Dr. Boice or me, or by both of us, and they all received practically the same postoperative treatment.

If the diagnosis of acute or perforative appendicitis, with or without abscess, is made with reasonable certainty, operation is performed immediately. A McBurney incision is made, varying it slightly as the appendix appears to be high or low. I am accustomed to work through this incision, and I believe that it is safer for drainage. The rest of the abdominal cavity is carefully walled off before the appendix or abscess is attacked. If the appendix is removed every effort is made to bury the stump to prevent fecal fistula. Only one persistent fistula requiring operation for closure has occurred in our practice. If the abscess is high, flank stab drainage is used and often drainage through a stab wound to the outer side of the incision to prevent breaking down of the wound which occasionally results in hernia. Only five such hernias have been noted. From three to five pieces of Penrose soft rubber are almost always used for drainage. These are placed carefully according to the site of the abscess or perforated appendix. If the pelvis has been invaded care is used to carry the drain to the bottom and leave it there on withdrawal of the carrier. Drains are shortened after the third day and are out by the seventh.

The question of removing the appendix in a

case of abscess should be left to the judgment of the surgeon who must take into consideration the necessary length of the operation, the amount of trauma, the danger of spreading infection, and, above all, the general condition of the patient. It is better to perform two operations than to perform one with the risk of causing death. There is little danger in draining an abscess, but the risk of removing the appendix is often five-fold if it does not readily come to hand. In practically all drainage cases a liberal amount of sodium chlorid solution is given subcutaneously during the operation, and in a fair number immediate gastric lavage. Patients are turned on the right side and the head of the bed is elevated only if the abscess is in the pelvis.

Post-operative treatment consists of hypodermoclysis, usually from 2500 to 5000 c. c. in twenty-four hours, liberal amounts of morphin every four hours, proctoclysis of soda, or glucose or sodium chlorid solution, if the stump of the appendix has been buried satisfactorily. However, proctoclysis is used less than formerly, as the subcutaneous method of giving fluid is more certain and does not cause peristalsis, which is undesirable in the presence of peritonitis. Nothing is given by mouth for from twelve to twenty-four hours, then, small amounts of water. Gastric lavage is carried out if indicated, but it is now rarely necessary; the "coffee ground" vomitus has disappeared since the adoption of hypodermoclysis in all perforative cases. Liquid diet is allowed on the third or fourth day if vomiting has ceased. Enemas are begun usually at the end of forty-eight hours. During the last three years glucose and insulin have been given intravenously when indicated in desperate cases. Blood transfusions have not been given as it was thought they were of little benefit in perforative appendicitis without hemorrhage. Serious post-operative hemorrhage has not occurred except in one case of hemophilia; the patient survived without transfusion. Mercurochrome has been used intravenously in two cases without benefit. In four fatal cases secondary enterostomy was performed. A few patients lived on whom primary enterostomy was performed. I believe this to be a valuable procedure that should be used more frequently.

Acidosis develops rapidly in the case of children and certain adults, if there has been much vomiting, and sometimes alkalosis develops. Dehydration is associated. These complications are treated by the free use of sodium chlorid solution subcutaneously or intravenously, and glucose intravenously.

A review of several recent papers giving classifications and mortality rate shows a surprisingly large number of cases of general peritonitis in which the patients survived. In this series it was not possible to determine which patients had general peritonitis except when it was found at necropsy; in all probability all such patients died. There is often general abdominal rigidity in cases in which there is a large free exudate which is culturally negative. A clinical diagnosis of general peritonitis can rarely be made. Many patients who recovered must have had more or less local peritonitis but not general peritonitis. It is inconceivable that drainage of one side of the abdominal cavity would clear up such infection without leaving walled-off abscesses in other portions of the abdominal cavity even if the patients survived. In seven of the thirteen cases coming to necrospy from one to many abscesses were found throughout the abdomen.

Necropsy was performed in thirteen of the twenty-three fatal cases.

SUMMARY

(1) Immediate operation should be done upon all cases of acute perforative appendicitis, regardless of the length of time since the beginning of the attack.

(2) Subcutaneous administrations of saline solution should be started simultaneously with operation and kept up until the water balance has been reestablished.

(3) The appendix should not be removed in all abscess cases, surgical judgment to be the guide in those cases in which it is left.

(4) Proctoclysis should be used only in cases of very limited peritonitis.

(5) Fowler's position should be used only in cases where the pelvis has been invaded by infection.

(6) Abscess and peritonitis cases above the brim of the pelvis should be turned to the right side.

(7) Provision for free drainage should be made in all cases.

SUMMARY OF CASES COMING TO NECROPSY

Case 1.—(698) A white boy, aged seventeen, had been sick for 7 days, but became worse after a wrestling match. He was purged twice with calomel and three times with salts. He entered the hospital June 14 and died July 2. The diagnosis was undetermined on admission. He was observed for several days, and then transpleural drainage of abscess in the splenic region was carried out. Later an abscess in the region of the gall bladder was drained and a third abscess was opened in the left inguinal region. There was never

TABULATION

Post-operative mortality in 382 cases of acute perforative appendicitis with and without abscess

Acute perforative appendicitis	White						Colored		Total		
	Aged 12 or less		Aged 13 and 14		Adults						
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Per Cent
Appendix removed	21	3	5		122	3	24		172	6	3.5
Abscess: appendix removed	23		8		66	5	30	3	127	8	6.25
Abscess: appendix not removed	18	1	3	1	28	1	34	6	83	9	10.8
Total	62*	4	16*	1	216	9	88	9	382	23	6.0

*If considered as in paper published in Southern Medicine and Surgery, 1925, lxxxvii, this figure would be 99. The discrepancy may be accounted for by the cases of acute gangrenous non-perforative appendicitis in which drainage was instituted, for fear of contamination, by simple drainage through the abdominal wall.

any symptom suggestive of appendicitis. Necropsy showed multiple abscesses secondary to the ruptured tip of a long pelvic appendix.

Case 2.—(4863) A white girl, aged sixteen was admitted November 9, 1919, with a large pelvic abscess of the appendix of four days' duration. The abscess was drained, and the concretion removed, with recovery. On the second admission November 20, 1920, there was another abscess in the left tubo-ovarian region. Leucocytes numbered 34,000. December 1, the appendix and the left tube and ovary were removed. The patient died four days later. Necropsy revealed general peritonitis.

Case 3.—(5017) A white boy, aged twelve, while convalescing from an attack of mumps, had abdominal symptoms for four days and was purged with calomel. At operation a large abscess was found around the head of the cecum extending into the pelvis; there was rather extensive peritonitis. The leucocytes numbered 19,200 and the temperature was 103.° The patient was admitted January 21, 1921, and died January 28, 1921. Necropsy showed abscess in the pelvis and numerous small pockets of pus throughout the left side of the abdomen between matted coils of intestines.

Case 4.—(6809) A white boy, aged nineteen, had been sick for three days, and had been purged with calomel and salts. The leucocytes numbered 16,600, the temperature was 102.6,° and the pulse 112. Operation was performed immediately after admission. A small appendiceal abscess was found beneath and attached to the liver. The appendix was removed. A subdiaphragmatic abscess developed, and repeated aspirations for pus were performed. A small median line epigastric incision was made for an exploratory operation under local anesthesia; a digital search for pus was made on the upper surface of the liver; no pus was found, but dense adhesions were encountered. The patient died forty-one days after the first operation. At necropsy a subdiaphragmatic abscess was found, extending below the liver and perforating the gall bladder; there was no peritonitis; there was pneumonia of the right lower lobe.

Case 5.—(9358) A white boy, aged seven, had been ill for two days when seen a day before admission. The temperature was 101° and he had diarrhea. On admission the temperature was 102° and the pulse 140. Leucocytes numbered 20,000. Appendectomy and drainage were performed immediately through a McBurney incision. The rectal temperature on the operating table was 105.° The post-operative diagnosis was acute gangrenous perforative appendicitis with extensive peritonitis. Enterostomy was performed on the third day, and the patient died on the sixth day. Necropsy revealed general peritonitis with pockets of pus under the liver and stomach, and in the pelvis. The small intestine was almost gangrenous in areas.

Case 6.—(9562)—A white woman, aged fifty-four, had been ill for ten days, and had been purged frequently. The leucocytes numbered 24,000, the temperature was 101,° and the pulse 116. Operation was performed immediately after admission. An appendiceal abscess was found and the appendix was removed. Four days later enterostomy was performed because of symptoms of obstruction. The patient died two days later. Necropsy revealed an abscess with intact wall, but no peritonitis. The cause of death was myocarditis, nephritis and possible septicemia.

Case 7.—(11136) The patient was a colored man, aged twenty-one, had been ill for seven days before admission, and had been purged. The temperature was 101,° and the pulse 120, and the leucocytes numbered 7,400. The patient was admitted July 21, 1925, and operation was performed immediately. Death occurred thirteen hours later. The post-operative diagnosis was general peritonitis. The appendix had sloughed. The stump was not buried. There was much pus in the pelvis and there were two round hard concretions. Necropsy revealed pockets of pus throughout the abdomen. The stump of the appendix was not leaking.

Case 8.—(10871) A colored boy, aged fourteen, had been sick for five days and had vomited for two days before admission. Salts and castor oil had been administered. The child was admitted May 29, 1925, and died June 4, 1925. The temperature was 100.4° and the pulse 100. The leucocytes numbered 16,400. Immediate operation was performed through a McBurney incision. A large appendiceal abscess was found extending into the pelvis, and the appendix was not removed. The temperature climbed continuously to 105.5.° June 2 a blood culture was positive for motile bacilli. At necropsy an abscess pocket was found which was well walled off. There was no pus in the cul de sac, no obstruction, and no peritonitis. The cause of death was septicemia.

Case 9.—(10474) A colored boy, aged fifteen, had been ill for three days. It was not known whether or

not he had been purged. The temperature was 102.2,° the pulse 120, and the leucocytes numbered 17,000. He was admitted March 4, 1925, and operation was performed four days later. A large appendiceal abscess was found in the pelvis. The appendix was not removed. Enterostomy was performed March 11 because of symptoms of obstruction. Death occurred three days later. Necropsy revealed bilateral pneumonia, pericarditis and pelvic peritonitis. There was no evidence of peritonitis in the upper part of the abdomen.

Case 10.—(8215) A colored man, aged about eighty, had been ill for five days, and had been repeatedly purged with castor oil. The leucocytes numbered 24,000, the temperature was 98.6,° and the pulse was 88. He was admitted August 9, 1923 and operated upon the next day. A large abscess was found and the appendix was not removed. Post-operative obstruction developed and enterostomy was performed under local anesthesia three days later. Death occurred one day later. Necrospy revealed the enterostomy opening three and a half feet above the ileocecal valve, no peritonitis, and the appendix ruptured. Death was probably due to senility and septicemia.

Case 11.—(9586) A colored woman, aged forty-nine, had been ill for ten days, and it was not known whether she had been purged. The leucocytes numbered 12,200, the temperature was 100.4,° and the pulse 96. She was admitted August 3, 1924. Operation was performed the next day and death occurred two days later. A moderate-sized appendiceal abscess was found and the appendix was not removed. The temperature rose to 105.4° and the pulse to 140 at death. Necropsy showed that the abscess was draining satisfactorily, and that the appendix was large, acutely inflamed, and perforated, and contained three concretions. There were large uterine fibromyomas. There was nothing in the abdomen to which death could be attributed. Death was probably due to septicemia, myocarditis, and fatty degeneration of the heart.

Case 12.—(492) A colored boy, aged fifteen, had been ill for seven days and had been purged daily, sometimes three times a day. He was admitted December 8, 1915. The temperature was 100.1° and the pulse 130. Diffuse peritonitis secondary to perforative appendicitis was diagnosed. Operation was performed under anesthesia through a McBurney incision; drainage was instituted and the appendix was not removed. About a quart of thin, foul-smelling colon pus under pressure escaped. Necropsy revealed extensive diffuse peritonitis with multiple abscesses secondary to the ruptured appendix.

Case 13.—(4936) A colored boy, aged eighteen, had been ill for two days and had been drastically purged. He was practically anuric. The urine contained albumin graded 3, pus, hyaline and granular casts, and blood graded 1. He was admitted December 19, 1920, and operation was performed immediately. The appendix was found to be acutely inflamed and perforated, and there was rather extensive peritonitis with free pus in the pelvis. About two-thirds of the appendix had sloughed. The appendix was removed. The patient died eight days after the operation. Necropsy revealed considerable free seropurulent fluid and generalized adhesions. The diagnosis was general peritonitis involving the upper and lower portions of the abdomen.

Discussion follows paper of Dr. Ogden, page 629.

SURGICAL DRAINAGE*

By M. D. Ogden, M.D.
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To paraphrase a saying of the late John B. Murphy, it is about time to discuss again the question of surgical drainage.

Twenty-two years ago Yates[1] carefully reviewed the subject of peritoneal drainage from the time of Hippocrates and showed conclusively by original experiments that (1) it is impossible to drain the general peritoneal cavity; (2) drains with smooth surfaces are more effective than drains with rough surfaces; (3) pus will not drain through gauze.

Omitting consideration of the pleural cavities and the various hollow organs such as the intestines, urinary bladder and gall bladder, a survey of the present-day literature on the drainage of infected wounds shows no uniformity of practice among surgeons and, in most articles, the mention of the material used is quite casual.

Drainage tubes of rubber are often used, some large, some small, some hard and some soft, and most of them have perforations at several points to allow the ingress of the wound secretion, the assumption being that the discharge reaches the surface of the wound through the lumen of the tube.

For small wounds, many writers mention the use of silkworm-gut, multiple strands of which are used simply as leads to guide the secretions to the surface.

Rubber tissue and rubber dam seem to be gaining in popularity and one finds increasing mention of these materials rolled into wicks without gauze. Robinson[2] advises that this rubber wick be perforated at intervals with a punch to allow the ingress of fluid, which is then removed by capillarity between the folds of tissue.

It is my impression, however, from actual observation and from scanning the current literature, that the cigarette drain is perhaps the most popular of all materials at the present time. The cigarette drain is a wick of gauze about which is rolled rubber dam or tissue. Directions for its manufacture vary. Some advise that a tuft of gauze be allowed to protrude from the end, others that it be cut off smoothly and still others

*Read in Section on Surgery, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

that the rubber covering be perforated at intervals.

Glass tubes are seldom mentioned nowadays, but the admonition is still occasionally found to insert a small wick of gauze for drainage.

Without reviewing minutely the pathology of inflammation, it will be profitable to outline here some of the essential phenomena which are factors in the present problem.

The response of tisssues to a bacterial injury consists, among other things, of the outpouring of tissue fluids known variously as lymph, plasma and otherwise, and the migration of various wandering cells to the injured area. If by lymph we mean the fluid contained within the lymphatic system, the term plasma would probably be more appropriate as a designation for this inflammatory fluid, which is generally regarded as passing through the walls of the dilated blood capillaries in the vicinity and from the tissue spaces. The lymph vessels do not open directly into the tissue spaces by means of stomata, as was once taught, but the system is a closed one, lined completely by endothelium and there is no scientific evidence that a reversal of the lymph flow occurs at any time, due either to inflammation or to the stimulus of introduced foreign bodies.

The blood plasma, under normal conditions, makes its way through the thin walls of the capillaries, diffuses through the tissues and into the lymph vessels, and this flow may be altered by the changes in capillary pressure or in the composition of the blood or by pathological changes in the tissues, thus disturbing the normal osmotic currents.

An abscess is an inflammatory area in which the exudate, consisting usually of plasma, fibrin, necrotic tissue and bacteria, is confined. With the release of the pressure, the contents of the abscess are extruded and healing progresses after the usual manner in inflammations by the formation of granulation tissue in the abscess wall, with the gradual obliteration of the cavity and with ultimate replacement of the granulation tissue by cicatricial tissue.

Following the opening of the abscess and the release of pressure, there then ensues a continuous flow of fluid containing tissue cells, pus and enzymes. The solid necrotic tissue liquefies under the influence of enzymes, the source of which it is not necessary to discuss here, and is discharged from the wound by reason of the contraction of the cavity incident to healing, by muscular action in some parts of the body and by the *vis a tergo* of the oncoming fluid from the tissue lining the cavity wall. Reduced to its smallest terms, the only function of a drainage material should be to act as a lead to guide these fluids to the outside and to maintain an opening until healing takes place so that the abscess contents cannot reaccumulate under pressure. Nature has provided stimuli in the form of fibrin, necrotic tissue and bacteria to cause sufficient secretion of this wound plasma and it is not necessary to introduce into the wound any irritative material such as gauze for the purpose of increasing this flow. To do so might well be compared to the application of a mustard plaster to a burn.

The ideal drainage material should then be one which is non-irritating, which provides an unobstructed passage to the outside and which by capillarity or suction removes the wound secretions. Such a material has not yet been found.

Rubber Tubes.—The lumen and perforations of rubber tubes are blocked very readily by granulation tissue and omentum and, after a little while, drain practically not at all through the lumen but act as encapsulated solid foreign bodies, around the periphery of which the abscess fluid finds its way to the surface. In addition to this there is the well-recognized danger that a stiff tube, in contact with the intestinal wall is very liable to produce perforation and fecal fistula. The same ill effects of pressure of rubber tubing are recognized when it is placed against large blood vessels, and necrosis of the common iliac artery has been caused by a tube inserted down to the ureter.

Gauze.—When a wick of gauze is inserted into a suppurating cavity, it acts as a drain by capillarity for the first few hours only. After that time the interstices become clogged and then it acts as a drain in the same manner as non-absorbent material, with several marked disadvantages. In the first place, it is holding in its meshes a large quantity of infective material and is a favorable field for bacterial proliferation. It even acts occasionally as a plug causing the abscess contents to reaccumulate under pressure, especially when granulations have grown into it, though this is not the case when the cigarette drain is used; and again, it is an irritative foreign body introduced into a wound that is attempting to heal and which is already sufficiently stimulated to this healing by the presence of fibrin, necrotic tissue and bacteria.

A gauze wick, with or without a rubber cov-

ering, then has the same drainage capacity as a solid cylinder of the same dimensions, that is, it serves solely as a guide to direct the flow of purulent material to the outside by keeping the wound edges apart and its capacity as a drain varies directly as its perimeter.

If gauze were the proper material to introduce into an appendiceal abscess, it should also be the proper material to use as a precautionary drain introduced down to a ligated cystic duct following cholecystectomy or to the suture line of a colon resection, but I do not know of its being so employed.

Eastman[3] utilizes the deleterious effects of gauze on tissue by introducing a tufted cigarette drain, extraperitoneally to an appendiceal abscess, leaving it in contact with the abscess wall which, after forty-eight hours, ruptures and the abscess is evacuated alongside the drain.

When Carrel[4] was developing his technic for the sterilization of wounds he soon found that it was impractical to interpose gauze between his tubes and the wound tissue as the gauze did not allow the Dakin solution to penetrate it after the first few hours. In his final technic, therefore, the tubes are laid directly upon the wound and the gauze upon the tubes.

It might be contended that too much space is here devoted to the condemnation of gauze as a drain, inasmuch as its popularity as such has greatly diminished with the years. However, gauze still has some earnest advocates and Kennedy[5] even goes so far in defense of his method as to say that the discarding of gauze as a drain is due to the use of rubber gloves in surgery, that the gloves so impair the tactile sense that infectious foci cannot be removed from the abdomen. I know that a certain number of men follow his teaching.

Payne[6] has written one of the most rational articles on drainage in recent years and advocates the use either of the cigarette drain or of rolled rubber tissue alone. When sloughing of tissue or fecal fistula occurs he removes the drains and replaces them by clean ones. He advises gentle irrigation, under very low pressure through a small soft catheter around the drain in those cases having thick solid discharges, and he also believes that drainage takes place about rather than through the drainage material.

I abandoned gauze and cigarette drains many years ago, replacing them with rolled rubber tissue. Following the introduction of Carrel's technic with Dakin's solution, I often introduced with the rubber tissue a Carrel tube, which led to the development of the present device.

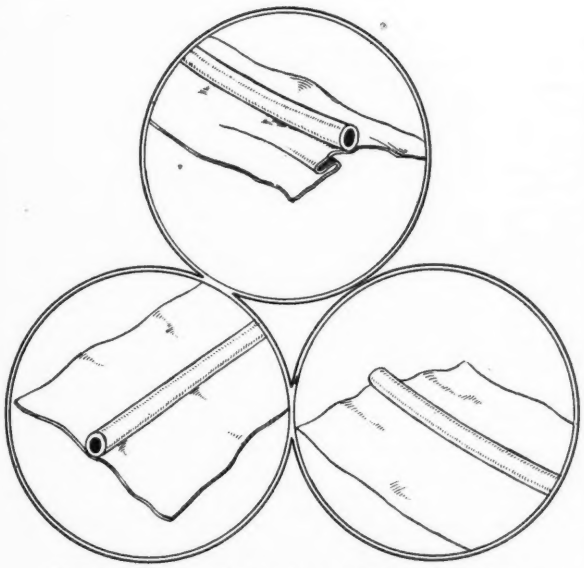
A strip of rubber dam 4 cm. wide is attached at its center to the side of a soft rubber tubing 5 mm. in diameter by rubber cement. This forms a soft rubber tube with two attached fins, each 2 cm. wide, and gives a total perimeter of 9 cm. to the finished drainage tube. This is ample for all but the most extensive wounds and by trimming off some of the fin it can be adapted to smaller wounds.

To illustrate its use in a case of suppurative appendicitis: the usual incision is made into the abscess cavity and the appendix removed, or not, according to the judgment of the operator. Then a section of this finned tube is introduced to the most dependent part of the abscess cavity from which it extends three or four inches beyond the skin surface. The wound is sutured with the exception of an adequate drainage aperture through which this tube protrudes and is dressed with gauze wet with Dakin's solution. The wound is allowed to remain undisturbed for forty-eight hours at which time the dressings are removed, when they will be found to have absorbed a varying amount of purulent secretion.

Irrigation with Dakin's solution through the tube either at two-hour intervals or with a continuous drip is then begun (the forty-eight-hour interval is to allow the circumscribing adhesions to become firm).

The Dakin's solution passes in through the tube to the depth of the wound, rises outside the tube with the wound secretion and is conducted by means of the fins to the surface where it is taken up by the dressings. This tube is, of course, not adapted to the drainage of hollow organs nor of the pleural cavity where a tight joint is desirable, as its purpose is to prevent the joint from becoming tight, but it is adapted to the drainage of practically all other parts of the body, such as perirenal, pelvic, appendiceal, osteomyelitic and other abscesses or conditions where rubber tissue, rubber tube or cigarette drains are now used.

The tube itself is not a drainage tube but is an irrigation tube with the advantage that the irrigation is retrograde, which is to say that it is in the same direction as the normal flow of wound discharge. The fins, of course, function only as drains and rubber dam is the least irritative material I have been able to find with the possible exception of paraffin which does not possess the requisite tensile strength



Carrel tubing cemented to strip of dental rubber dam.

for this purpose. Rubber dam is superior to rubber tissue as its greater strength lessens the chances of fragments being left behind in the wound upon withdrawal of the drain.

Obviously one would not use this drain in very small wounds, nor as a precautionary drain, nor in situations where a small strip of rubber dam alone suffices; but it can be used wherever a drain of this size is desired, even though irrigation is not employed.

This drain is easily manufactured by any operating room force, along with the sponges and other supplies. A strip of Carrel tubing and a strip of rubber dam two inches wide are roughened with emery paper, spread with a thin line of rubber cement and glued together. It is then allowed to dry, when it is boiled or autoclaved and stored in antiseptic solution, usually Harrington's, whence it is removed as needed, washed in sterile water and handed to the surgeon. The tip of the tube is placed in the deepest part of the abscess cavity and led out through the incision which is closed down to the tube, leaving sufficient room to avoid constriction. As many tubes can be used as there are cavities to drain, though it is occasionally possible to lead the tube from one cavity through another to the outside. It is left in as long as the judgment of the surgeon dictates. It is possible in this way to obtain adequate drainage and at the same time avoid larger openings in the incision with their liability to subsequent hernia.

By its use with hypochlorite solution, wound healing is hastened, disagreeable odors are minimized or abolished, reinfection from the surface is obviated as the current is ever outward and routine dressings are not nearly so irksome to patient or surgeon. It is not a perfect drain but is, I believe, superior to the cigarette and to rolled rubber tissue alone.

SUMMARY

Abscesses contain necrotic tissue, fibrin and bacteria which are sufficient stimuli for the production of wound secretion. Irritant foreign bodies introduced for this purpose, are therefore unnecessary and harmful.

Rubber tubes are unsuitable drainage materials and often produce ill effects by pressure.

Gauze drains only slightly by capillarity, harbors infective material, is irritating, and after the first few hours drains only around its periphery.

Cigarette drains are less objectionable than gauze alone, but are far from ideal.

Rubber dam alone is the best available drainage material at this time and its use when combined with a small retrograde irrigation tube affords the most rational method of hastening the healing of infected wounds.

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DISCUSSION (*Abstract*)

Papers of Dr. Willis and Dr. Ogden

Dr. Floyd W. McRae, Atlanta, Ga.—If we analyze Dr. Willis' low mortality we may find several factors which are important. First, the incision which he adapts to the most obvious location of the appendix, and the gentleness with which he handles the structures after the abdomen is opened. The less intra-abdominal manipulation and the less trauma to the peritoneum the better, and this is probably a great fac-

tor in the healing, and is best obtained by the long right rectus incision. When we find a staphylococcic infection we are apt to feel better than if we open an abdomen and find very little drainage, with a gangrenous appendix. This is indicative of a streptococcic infection, in which the mortality is very high, perhaps 100 per cent. Perhaps the question of inversion or non-inversion of the stump is a moot one, and depends largely upon the judgment of the operator. I have noted hemorrhage in a few instances caused by a false attempt to remove tympanitis. Gas may be forced into the cecum with even low pressure because of blowing out the stump of the appendix.

Dr. Willis did not mention one thing that has given me trouble, and that is subphrenic abscess. If this is sought for perhaps the mortality can be still further decreased. One dictum should be remembered: that purgation spells perforation.

Fluids post-operatively are very important. Hypodermoclysis is far preferable, in my opinion, because there is very little absorption from proctoclysis.

Dr. William Britt Burns, Memphis, Tenn.—I have gone over the whole question of drainage as far back as the time when we used to put in coffer dams and leave them in, and several yards of gauze and leave that in, and give an anesthetic later and take it out. I found no practical help in that, and only wish to say that I think Dr. Ogden has covered in a little sentence the opinion on the matter of drainage, and in the matter of absorption by the peritoneum. There is some considerable confusion yet as to the matter of absorption, and as to the lanes of fluids, how they get in and how they get out, and how much this is influenced by osmosis and the ordinary dialysis, and I am forced to the position of getting down to a simple, practical method of drainage. Dr. Ogden is perfectly right in the use of drainage material that does not adhere. The rubber dam is probably among the best agents. I have been in the habit of using silkworm gut in a large number of strands, which has seemed to serve me just as well. When there is an abdomen filled with pus I have used the ordinary stab drain in the flank. I think the best way to provide this is to put a drain through the back and a drain through the stab wound in front, and calculate the amount of drainage in the two pads, in front and behind. Ninety per cent of the fluid goes into the back pad.

I wish to refer to the question of pelvic abscess from appendices and the matter of diagnosis of appendices that are found in the pelvis. The principal thing that will help in the diagnosis of such a case is the bladder symptoms. I have been able to make the diagnosis of appendicitis by the general symptoms and by the general bladder symptoms of frequent irritation, and effort to urinate.

Dr. Wallace Frank, Louisville, Ky.—As Dr. Ogden says, this method serves two purposes; first, drainage, and then it furnishes a pathway for putting irrigating fluids into the wound. Drainage is introduced for one of three purposes: (1) to relieve pressure; (2) to establish a sinus by which infectious materials can escape; and (3) to wall off infected areas. Drainage of the general abdominal cavity is impossible. The infected material is walled off in a few hours and we are draining just a part of it. However, there are in the abdomen definite spaces in which there is room for fluids to accumulate, in the renal fossa, in the ileac fossa, and in the pelvis, and in those cases we think drainage should be instituted in all five areas.

Regarding the material used, to our mind up to this time the best was the cigarette drain. Gauze is of advantage for a coffer dam drain to wall off severely infected areas from the pelvic cavity. We have all seen bad infections where it was necessary to open the abscess. It is rough, it stimulates the out-pouring of fluids to combat the infection, and it hastens the walling off of that area from the peritoneum. Gauze has another place in the abdomen, and that is as a tampon. Dr. Ogden mentioned in the paper that gauze is not used in the cystic duct. Occasionally one sees following cholecystectomy great oozing from the bed. In such a case gauze can be used as a tampon to advantage. I have never seen biliary leakage following the use of the gauze tampon in this way.

There are only two places to use tubes either in a localized collection or in a cavity. Otherwise, the hard rubber tube has no place in the abdomen. It is likely to produce a fistula into the intestine and necrosis in the large vessels.

Regarding the distum now largely preached of "when in doubt do not drain," I must say that I agree with Payne in his statement that such teaching has disadvantage. In the practice of men of wide experience there are but few occasions upon which there is any question of whether to drain or not, but in the hands of men who do surgery all over the country this question does arise, and I think when in doubt it is much better to drain. We can save the lives of far more individuals by so doing than by leaving out the drainage.

Dr. Alton Ochsner, New Orleans, La.—I wish to make a plea for conservative surgery. I have seen the radical surgery used in Professor Clairmont's clinic in Zurich. I cannot cite figures, but in Clairmont's clinic in 10 per cent of cases a subphrenic abscess developed. The reason for this was that the patients were operated upon in the acute stage of general peritonitis. This has also been emphasized in the report of the Middlesex Hospital, where before the introduction of conservative methods there was a high incidence of subphrenic abscess, but following the adoption of conservatism a great drop occurred in the mortality. Death following a suppurative appendicitis is due either to peritonitis or septicemia. If cases of acute appendicitis are seen twenty-four to thirty-six hours after the onset, and localizing signs are present, it is much better to wait until complete walling-off has occurred. If the case is operated in the face of fresh adhesions, it is necessary to break down the dam which nature has produced, in an attempt to wall off the infection. A high mortality will result, due to a general peritonitis. I happened to be in Poole's clinic one day three years ago and was asked my opinion regarding conservative surgery of appendicitis. Dr. Poole felt that all appendicitis was immediately operable. I attended their staff meeting, where the deaths which had occurred during the week were considered. In one case a patient had been admitted with a history of symptoms of appendicitis for four days. Operation was performed, a definite abscess was

found, and the appendix was removed. The patient died a few days later of general peritonitis. Dr. Poole admitted that if they had waited before operating the death would probably not have occurred. I feel confident that the mortality will be less in the late cases if conservative methods are used.

Dr. S. L. Ledbetter, Jr., Birmingham, Ala.—I am in accord with everything Dr. Willis has said regarding the advantages of the McBurney incision.

One of the causes of the high mortality in acute appendicitis is post-operative obstructions, both immediate and remote. The McBurney incision allows for a much better placing of the drains than other incisions, and we have practically no post-operative obstruction either immediate or remote. The drains should always be placed lateral to the intestinal loops next to the pelvic and ileac wall and this cannot be done through a rectus incision.

Regarding drainage, I am reminded of the remarks of the late Dr. W. S. Halsted, that we drain for several things: first, in the presence of necrotic material; secondly, in the presence of oozing; and thirdly, where there is danger of leakage from a hollow viscus. Therefore, with a gangrenous appendix that has not perforated I never use a drain. Until two years ago I always used the rubber covered gauze cigarette drain. Recently I have occasionally used a small soft rubber stick drain which is very good. The cross section of this drain is x-shaped and around this several rolls of rubber dam are placed. That allows drainage along the folds of the rubber dam and in the triangular spaces of the drain. This is quite soft and does no damage. We remove one in two or three days, thus giving a larger drainage tract around the remaining ones.

Dr. Willis (closing).—A young school teacher last winter was seized with acute appendicitis and she did not reach the hospital for forty-eight hours. Her temperature was 102°, pulse 160, and she was very ill. I said "if there ever was a case in which to delay this is the one." I waited, and four days later I had to do an enterostomy for obstruction. This young woman was given hypodermoclysis and nothing by mouth, but still she was in desperate straights. After three months her condition had improved slightly. Then we found the appendix attached to the anterior abdominal wall, under McBurney's point. It would have been the easiest thing in the world to remove or drain that appendix primarily and we could have saved the girl months of suffering.

I feel that Dr. Ogden's drain is better than the simple Penrose. It will enable us to drain the bottom of the cavity and irrigate at the same time. We use five pieces of soft Penrose drainage material, feeling that the multiple pieces give an opportunity for drainage between them. It does this very well, but as you know the pocket walls off very readily and the balance of the abdominal cavity is not drained.

I cannot close without calling attention to general peritonitis. I do not believe we are having so many cases as are recorded in the literature.

Dr. Ogden (closing).—I have tried to use the Penrose drain for irrigating purposes but abandoned it because the walls were so thin that it buckled up and pushed itself out of the wound. With this kind of a drain stab wounds are rarely necessary; for if the drain reaches the bottom of the wound the walls will fill up behind it very readily upon gradual withdrawal.

EVALUATION OF THE DIFFERENT FACTORS IN SYPHILITIC THERAPY*

By THOMAS W. MURRELL, M.D.
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Dr. Durant in his "History of Philosophy," considering the work of Aristotle, and the virtue and defects of the Greek mentality, states:

"So Greek philosophy leaped on to heights unreached again, while Greek science limped behind. Our modern danger is precisely opposite; inductive data fall upon us from all sides like the lava of Vesuvius; we suffocate with uno-ordinated facts; our minds are overwhelmed with sciences breeding and multiplying into specialistic chaos for want of synthetic thought and a unifying philosophy."

This so aptly describes the state of syphilitic therapy, that the writer feels it an excuse for presenting a paper to a scientific section, which is discursive rather than informatory.

As a man thinketh has ever been the measure of his acts, and comparing progress we have to consider the thinking of other days as much as the work done. Syphilis became an historical reality in the immediate post-Columbian period and the idea of the specific dominated all therapy for over two hundred years. It is natural that it should have been so for the most educated was very superstitious and one drug after another was employed in this way. Sasaparilla and guaiac were among the first, and as late as 1897, in one of the standard materia medicas, guaiac was still so classed for its supposed action on syphilis. Mercury, when first used, was employed in this fashion. A patient was given a course of mercury by rubbing with the intention of producing a severe salivation. This was supposed to be a cure, but of course, candid observation showed it to be untrue. There was apparently nothing to do but to re-salivate, but this was a procedure too severe and harmful to be permitted many times and so the need was for a continued treatment.

Von Sweitman, of Vienna, met this by administering bichlorid of mercury by mouth. This was around 1750 and the next one hundred and sixty years was given over to the development of the idea of the continued treatment. This was expressed in giving over a period of time as much mercury as the patient could handle without detriment. This was to be followed

*Read in Section on Dermatology and Syphilology, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

by a shorter period of cleaning up with the iodids. The classical time was three years of mercury and one year of iodid. Different forms of mercury had their advocates; the French with the proto-iodid, the English with calomel and mercury with chalk, the Germans holding to the inunction, but there was an unanimity of opinion as to the time of treatment, and this time factor is really the condensed therapeutic principle, which is the result of the thinking of master minds for a century and a half.

Now in 1910, the arsenicals, in a non-toxic form came to us, but it is nothing short of a tragedy in what guise they came. Harking back over the centuries the old specific idea came back into being. Dazzled by the vision of the therapeutic *sterilisans magna*, we cast aside the bitterly gained knowledge of the time factor and are only now emerging from the chaos, which this specific phantom produced.

The dividing line between the old and the new in syphilis is the year of 1905, when Schaudinn discovered the spirochete. This still remains a clean cut discovery with no false claims to blight it. In 1907 the serological discovery, commonly called the blood test, or the Wassermann reaction, came to us. This came as a test and was so accepted by profession and laity. But it was not a test, though the majority of the profession, and all laymen think of it as a test, in all that the name implies. Lastly came the arsphenamins in the evil garb of ancient superstition, and there is no telling the harm the false thoughts they aroused have caused.

There are four factors in therapy:

(1) The general physical condition of the patient.

(2) Drugs.

(3) Time over which drugs shall be used.

(4) Serology in diagnosis and guiding treatment.

There is no question as to the necessity of holding the patient to the highest level of health, so the first needs no discussion. The drugs used are known to all and the manner of their using is but a matter of technic and need not be considered here.

The time covered by treatment is bound to be a matter of vast importance. When the masters of many generations come to a conclusion, they are in the main right, and if the old time factor, which governed other days, is to be discarded, then a new time factor must be established. It may be one-half or one-fourth, the older measure, because of new and more powerful weapons but it still exists and every effort should be made to ascertain it and make it known.

The fourth factor is serological examination. To say the least, this procedure occupies a unique position. Nowhere in medicine are such momentious decisions made on evidence, which presents such indefinite credentials as the serum reaction. They are related in some way to syphilitic pathology, we yet do not know how, nor why. The reaction may be negative in the face of blatant syphilis and yet illogically when positive has risen to the court of the last resort. When the infection is new this reaction concides fairly closely with the progress of the disease and rises and falls with other evidences, so when the diagnosis is made, it becomes a fair guide to treatment. Let time play its part and the Wassermann loses all sense of synchronization. In such a case, a late case, all symptoms may clear up under treatment; a patient may be at a high level of physical excellence by all other means of medical observation, yet the four-plus report comes in with deadly monotony, perhaps finally disappearing.

Then come the cases called Wassermann-fast, whatever that means. Certain it is, there are thousands of such individuals and their plight demands something more than they are getting. Obey every mandate of education and good sense, and you still get the positive report. In Virginia, we have a law, where a party being convicted of immoral, or disreputable conduct, and sentenced to detention, cannot be released as long as he has demonstrable syphilis, and this means a positive Wassermann. At a certain home for delinquent girls, I know of a case, who has received over fifty injections of the arsenicals, with numberless mercurials and iodids, who still runs a consistent positive Wassermann. There is no other sign or stigma of the disease, but because she gets this report she is still confined in the institution. If the writer's experience be any guide, unless the laws are changed, she is likely to become a permanent inmate.

But are cases such as this necessarily syphilitic? Are there cases which are incurable? Must we condemn patients to endless treatment on

only one factor in therapy, when all other factors have been obeyed? Why not weigh the evidence and view it from another angle?

Not until the relationship of immunity to the Wassermann reaction is established shall we be able to see clearly what it all means. Acquired immunity and hereditary immunity are accepted as facts and expressed as laws. Where there is an exception, it will occur in the acquired type rather than the hereditary type and these hereditary cases particularly late of all the varieties of syphilis seem to have the most resistant Wassermanns. In other words, in a class where the Wassermann remains most immovable under treatment, there is the highest type of immunity.

It was the established custom of twenty-five years age to avoid treating chancres, and to withhold treatment until the secondaries developed. We were told to do this because a little treatment might mask syphilis and such cases would therefore neglect themselves and such neglect would lead on to neural syphilis. It was further taught that neural syphilis was much more apt to develop from a mild case than a severe case, but usually the patient was blamed for neglecting treatment. May not these men have seen right and interpreted wrong? May not insufficient early treatment have interfered with immunity and thus allowed infection of the higher tissues? It has been noted that patients with active syphilis, whose Wassermanns become quickly negative, do not do well; and patients, who have active syphilis with negative Wassermanns are peculiarly prone to relapses with clinical symptoms. The large percentage of nerve and heart syphilis with the negative Wassermann is a commonplace. Surely these facts must have some explanation other than specific tissues susceptibility, or anatomical separation. And lastly, and it takes much nerve to propose the question; may not the Wassermann-fast case be actually an intensive degree of immunity? One thing is sure, positive serum and contagion are not the same. Hutcheson states that he has never seen contagion from a gumma, and the use of positive bloods in surgical transfusions with no resultant harm has lately been reported.

The negative Wassermann early became a bad citizen. We teach that no reliance may be placed upon it, yet publish case reports, almost naive in this, that on becoming sero-negative the case was discharged. A discredited agent is called in to prove good work, just as in other circumstances its blood brother stalks in to disprove work equally good. Yet this serological reaction is too wonderful a compass to do without when sailing syphilitic seas, and the comparison is good in this; there are normal variations to the compass, and it sometimes goes entirely wrong.

While no one can say with definiteness the actual meaning of the Wassermann, conversations with many men in all lines of work show the majority feel that even if it is not an expression of immunity it surely is in some way connected with the defense reactions of the body.

Complement-fixation in other diseases such as pneumonia and arthritis is accepted as good news showing resistance. If this be so, it is not strange that a similar process in another disease should be regarded as bad news.

If syphilis is being poorly treated by the profession at large it is merely because of wrong thinking. The average medical man is an honorable man and does his duty as he sees it and syphilis will be properly treated when this average physician thinks properly.

Twenty-five years ago this average physician had one stabilizing thought and this was the time factor. He abandoned this and has substituted serological reaction; has in other words substituted the unknown for the known.

Undoubtedly most of the trouble with the rank and file has come from putting a quantitative meaning into the serological phenomenon. Even if this reaction be accepted as indisputable evidence of an abnormal condition, we have not the slightest idea of how abnormal the condition would be. Therefore if serology be the only guide, one is as justified in suspending treatment with a four-plus as with a two-plus. It would be a better plan to discard entirely these graduations, letting the report be a negative or positive, then reserving the right to interpret this report in correlation with other factors.

As to the heresies herein expressed, permit a quotation from Francis Bacon:

"If a man will begin with certainties he shall end in doubt; but if he will be content to begin in doubts, he shall end in certainties."

Discussion follows paper of Dr. Lord, page 643.

SYPHILITIC HEART DISEASE WITH FAILURE*†

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Both scientific and popular literature have contained much concerning heart disease in recent years. It is an established fact that heart disease is causing a relative and actual increase in morbidity and mortality in the United States.[1] Syphilis is a prominent etiological factor in cardiovascular disease. Cohn[2] states that 18 per cent is caused by syphilis. Wood, Jones and Kimbrough[3] found syphilis the cause in 11 per cent of three hundred cases in Virginia and 6.67 per cent of three hundred and twenty-three cases in Massachusetts. Stone and Vanzant[4] in a recent report from Texas found syphilis the etiological factor in 19.3 per cent of nine hundred and fifteen cases of heart disease. These figures probably do not reveal the actual total incidence of cardiovascular involvement in syphilitics. Also there is a notable amount of syphilitic disease not recognized.[5]

Since efficient prophylactic and therapeutic measures are now available, it is reasonable to hope that this portion of heart disease will be eliminated. Perhaps no other form of infection leading to heart disease is quite so definitely preventable as syphilis, and the non-infectious types of cardiac involvement are at present largely beyond control.

At the outset it was soon seen that the records in regard to early cardiovascular syphilis lacked elements of completeness. Present day clinical cardiology has not advanced sufficiently to determine early and minor abnormalities with accuracy. This investigation was limited to a clinical study of hospital cases of syphilitic heart disease with failure. By failure is meant any cardiac or aortic defect that gives rise to complaints by the patient at his usual level of activity, or that enforces any limitations of his normal demands.

This analysis is based on the study of fifty-six cases admitted to the University of Virginia Hospital between July, 1923 and July, 1927. All of the cases had heart failure and were diagnosed syphilis of the heart or aorta. Syphilis was considered the primary etiological factor; this was proved by autopsy in some of the cases but in most of them the diagnosis was based on clinical and laboratory data. The cases of heart disease showing what was considered an incidental syphilitic infection and those in which it was thought to be of secondary importance, were discarded. In addition to the usual medical investigation consisting of history, physical examination and routine laboratory methods, thirty-four patients had electrocardiograms, forty-two were x-rayed and eight were autopsied.

Of the fifty-six cases, thirty-nine (69.6 per cent) were negroes, and seventeen (30.4 per cent) were whites. The proportion of negro syphilitic patients to white syphilitic patients in the hospital and out-patient department was 3 to 2. From these figures it is estimated that syphilitic heart disease occurred one and one-half times as frequently in syphilitic negroes as in syphilitic whites. The average age of the negro patients in this group at admission was 45.8 years as compared with 52.6 years for the white patients. The greater incidence and the earlier occurrence in the negro indicates an increased susceptibility in this race to cardiovascular involvement from syphilis.

There were forty-nine male and seven female patients. Fourteen were farmers, twenty were laborers and the occupations of all the others required considerable physical exertion. The average age at admission was 47.7 years.

There was a positive history of a genital sore in twenty-five cases. The average interval in these from the initial infection to the onset of failure was twenty-two years. The shortest interval was eight years, the longest forty-one years.

Only one case gave a history of early cutaneous lesions. This would seem to confirm the idea expressed by Brown and Pearce[6] in their law of inverse porportion, that severe late visceral involvement occurs more often following mild early reactions. This finding is paralleled by the well substantiated observation that neurosyphilis is relatively less frequent in those patients who have had severe skin symptoms.[7] It is impossible at present to say whether this is due to different strains of organisms, or to an individual visceral susceptibility.

Tabulation of the initial symptoms gave the following results: dyspnea 29 (51 per cent),

*Read in the Section on Dermatology and Syphilology, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

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heart pain 10 (18 per cent), palpitation 10 (18 per cent), substernal pain 3 (5 per cent), weakness 3 (5 per cent), cough 2 (4 per cent), vertigo 2 (4 per cent), headache 1 (2 per cent), and dyspepsia 1 (2 per cent). Some showed more than one initial symptom. Other early symptoms were: edema 28 (50 per cent), dyspnea 14 (25 per cent), heart pain 12 (21 per cent), cough 12 (21 per cent), palpitation 11 (20 per cent), vertigo 10 (18 per cent), weakness 9 (16 per cent), substernal pain 4 (7 per cent), dyspepsia 3 (5 per cent), choking sensation 3 (4 per cent), nervousness 2 (4 per cent), sleep starts 2 (4 per cent), headaches, convulsions, insomnia, cyanosis, hoarseness and "buzzing sound in heart" 1 each.

Functionally twenty-five cases presented both congestive and anginal failure, thirty showed congestive failure alone and one anginal failure alone.

The abnormal structural findings, based on physical examinations, teleroentgenograms, fluoroscopic examinations, and autopsies were as follows: cardiac hypertrophy 51 (90 per cent), aortic regurgitation 33 (59 per cent), dilated aorta 31 (55 per cent), aneurysm of the aorta 18 (32 per cent), myocarditis 3 (5 per cent), and coronary occlusion 1 (2 per cent).

In three of the eight autopsies, marked fibrous and degenerative myocarditis was noted. These were the findings in routine post mortem examinations and it is probable that special pathological studies would have revealed other instances of specific myocardial changes. Cabot[8] in his recent book reports only ninety-two cases of syphilitic heart disease in a total of nineteen hundred and six necropsied heart cases. Warthin[9] in his seven hundred and fifty autopsies found what he considered positive evidence of syphilis in forty per cent of the cases and involvement of the heart in all of these. He says:

"the heart in every case showed lesions characteristic of spirochete localization; and in this organ more frequently than in any other has the spirochete itself been demonstrated by the Levaditi method."

He states that the striking difference between his findings and those of other pathologists is dependent upon the different pathologic criteria employed. Warthin's criteria are chiefly microscopic while the conclusions of others are based on macroscopic data. However, the argument relating to the pathological criteria may be decided, modern conceptions regarding the distribution, characteristics and behavior of the *Treponema pallidum* favor frequent myocardial involvement in all stages of syphilis.

Forty-nine of the fifty-six cases had positive blood Wassermann reactions, a percentage of 87.5. Of the remaining seven cases, syphilis was evidenced by other conclusive findings.

Twenty-six of this group had spinal fluid examinations. There were normal findings in fourteen of those tested: in twelve instances, or 46 per cent of those examined there was positive evidence of central nervous system involvement. The positive spinal fluid findings were the means of confirming syphilis as the etiological factor in some of the cases with negative history and negative blood Wassermann.

The average blood pressure in the thirty-three cases of aortic regurgitation was systolic 150, diastolic 60, pulse pressure 81. Cases without aortic regurgitation showed systolic 138, diastolic 80 and a pulse pressure of 58. The average blood pressure in all cases was systolic 145, diastolic 74, giving a pulse pressure of 71.

The thirty-four electrocardiographic tracings revealed nothing distinctive. Left axis deviation was present in most cases as might be expected in left cardiac hypertrophy. Five patients had partial or complete heart-block.

The forty-two roentgenographic examinations proved the value of this procedure in determining the structural abnormalities in the heart and aorta. Several cases with negative physical examination showed definite changes such as aneurysm or dilated aorta on x-ray examination. This method of examination was also valuable in differential diagnosis from an etiologic standpoint.

Forty-eight of the fifty-six cases gave no history of anti-syphilitic treatment. The remaining eight gave a history of "internal" treatment, probably mercury and iodids. Most of the cases in which a history of initial infection was obtained began before the modern era of anti-syphilitic treatment. The few cases that acquired their infection since the discovery of arsphenamin gave no history of intravenous treatment. It will be interesting to determine later whether or not modern anti-syphilitic therapy completely prevents late cardiovascular involvement. It is presumed with some assurance, based on clinical observations[10] and experimental findings[11] that late syphilis is preventable.

Twenty-one of the fifty-six cases are known to be dead, eighteen are known to be alive and the condition of seventeen is unknown. Of the eighteen alive, thirteen are improved and five are unimproved. In the group of twenty-one which are dead the average interval from the onset of failure to death was 3.2 years. The average interval since onset of failure in those alive and heard from is 4.1 years. There is undoubtedly a factor in certain of the cases, which accounts for the more rapid progression of the heart deficiency, but it has not been determined in this study.

SUMMARY

(1) Syphilis is an important etiological factor in heart disease especially in the Southern states. The prevention of syphilis will eliminate this group of cardiovascular disorders.

(2) The findings in fifty-six cases of syphilitic heart disease with failure are in part presented. Age, sex, race, occupation, early history, initial heart symptoms, clinical and laboratory results are considered.

(3) The negro race in this country is probably more susceptible to syphilitic heart disease than the white race.

(4) Only one in the group of fifty-six cases gave a history of early cutaneous lesions.

(5) Available clinical and experimental data indicates that modern anti-syphilitic treatment in the early stages will prevent late involvement of the heart and aorta.

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Discussion follows paper of Dr. Lord, page 643.

A STUDY OF WASSERMANN-FAST SYPHILIS*

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Whether there is a correlation between the so-called Wassermann-fast phenomenon and the existence of active syphilitic processes in patients who exhibit this phenomenon is a question which has never been fully answered. The present study was undertaken in the hope that an answer might be presented.

Prominence has been given to the desirability of arriving at a proper interpretation of Wassermann-fastness by clinicians seeking guidance in the treatment of such patients. It is obvious that any therapeutic attack depends for its justification on the presence of active syphilitic disease as the basis of the positive Wassermann reaction. If the blood Wassermann reaction may continue to be positive after healing of all lesions, and the complete destruction of all *Spirocheta pallida,* it is clear that no basis for treatment remains.

The question is therefore: what causal relationship, if any, exists between the tissue reactions associated with syphilitic disease and the positive Wassermann reaction?

The generally accepted facts which appear to bear on this question are the following: (1) the Wassermann reaction does not become positive until the appearance, or shortly subsequent to the appearance, of the primary lesion. (2) During the secondary stage of syphilis, when tissue reaction to the syphilitic virus is most widespread, the Wassermann is positive in at least 98 per cent of cases. And conversely, treatment during this stage causes the reversal of the reaction to negative, but almost always not until the disappearance of the lesions. (3) In syphilis of the experimental animal[1] the Wassermann becomes positive coincidentally with, or shortly after, the appearance of the chancre, and becomes negative coincidentally with its disappearance, although infection persists, whether the lesion be healed spontaneously, by treatment, or be excised.

These facts by themselves, while tending to support the view that a positive Wassermann is a product of the syphilitic disease process, cannot be interpreted as denying the hypothesis that

*Read in Section on Dermatology and Syphilology, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

TABLE I.

PATIENTS WITH PERSISTENT POSITIVE WASSERMANN REACTIONS
118 CASES

	59 Men							59 Women							
	Under 10	11-20	21-30	31-40	41-50	51-60	Over 60	Totals							
C. N. S.		2	8	12	6	3		31							
C. V.			5	6	7	3		21							
Bone			4	7	1	1		13							
Congenital	4	10	1					15							
Visceral			3	1				4							
Latent			1	1	3	2		7							
More than 1 lesion		7	4	5	5	4	2	27							
Totals	4	19	26	32	22	13	2	118							

cells which have acquired the property of causing the Wassermann reaction in the fluids of the body, may continue to cause a positive Wassermann after the disease process has been terminated and the infection eradicated.

A study of Wassermann-fast patients may throw light on this aspect of the question, if it can be shown that such patients still have foci of active syphilitic inflammation. Furthermore, if it can be shown that there is a class of syphilitic patients whose disease is practically invariably cured, with sterilization of the infection, and that in this class Wassermann-fastness is unknown, the above hypothesis becomes unnecessary.

There is no general agreement as to exactly what constitutes Wassermann-fastness. Stokes and Bushman,[2] in a somewhat similar study, selected those patients with persistently positive tests after a minimum of twelve injections of an arsphenamin and three months of mercury; Kaplan[3] and W. Fox[4] speak of the patient on whom "no amount of treatment will effect a reduction." We have designated as Wassermann-fast all patients who have had one year's regular and active treatment with persistently positive tests.

In the compilation of our data, the histories of all patients treated in the syphilis clinic of Johns Hopkins Hospital between October 1918 and January 1927 were reviewed. During this time there were approximately 8500 admissions. Of this number approximately 900 received regular treatment for a year or more. From these latter were selected all those cases which fulfilled the criterion of Wassermann-fastness as arbitrarily laid down above 118 in all.

The patients studied in this series have had routinely careful physical examinations on admission to the clinic; spinal punctures were performed in almost all cases; roentgenologic examinations and other tests which were deemed advisable were made. Their treatment was carefully supervised, they were re-examined at regular intervals, and finally, as many as could be brought back to the clinic were again examined by the writer. The routine continuous treatment was used. This comprises courses of an arsphenamine product, or tryparsamide alternating with either mercurial inunctions and potassium iodid by mouth, or with bismuth, usually bismuth salicylate, intramuscularly.

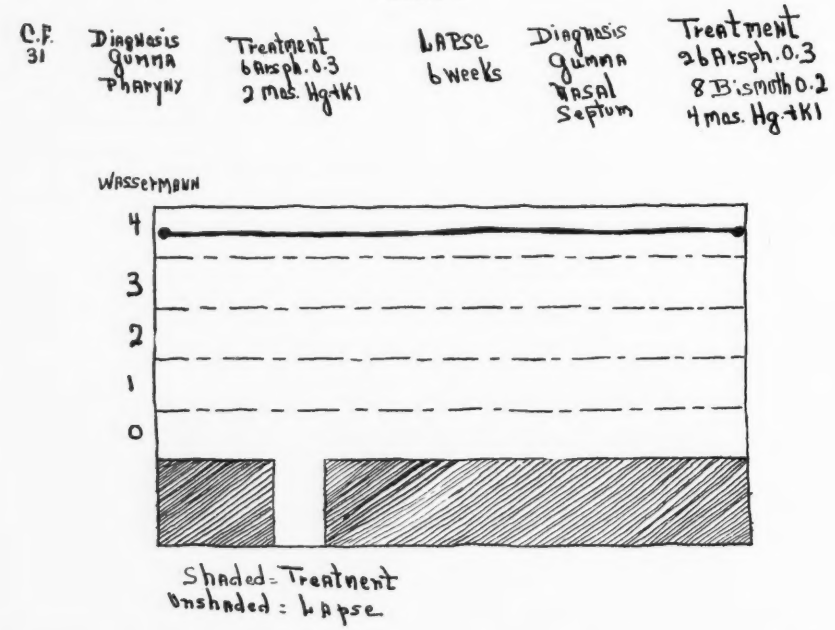
For the purpose of study the material is grouped according to age, sex and anatomical diagnosis (*Table 1*). Sex does not appear to be a factor. Some significance can be attached to age, although in a relative way rather than actually. It is expected of course that congenital cases will appear in the first and second decades largely, and in this clinic all such patients exhibit lesions of late syphilis. The manifestations of acquired syphilis presented by these patients for treatment do not appear until late in the course of the disease. The age of the patient therefore is of importance in so far as it relates to the probable duration of the disease from the age incidence of primary and secondary syphilis which in this clinic is highest in the second and third decades, especially in the second half of the second decade. In the table the incidence of Wassermann-fastness is highest in the third, fourth and fifth decades, the greatest number of patients being in the fourth decade. One may assume in this way that the duration of the disease in these patients has been from one to thirty or more years. That the duration is not less than one year, we know because in this clinic we have not observed a single instance of Wassermann fastness in primary and uncomplicated secondary syphilitic patients who have completed their treatment on the uninterrupted therapy scheme for one year after permanent reversal of the Wassermann. The significance attaching to

TABLE II

PROOF OF SYPHILIS

	Number	Percentage
Active Disease	56	47.5
Activity Equivocal	58	49
No evidence of Syphilis	4	3.5
Total	118	100

CHART I.



the age of the patient therefore is relative to chronicity of the disease.

In grouping these patients according to the anatomic diagnosis, congenital syphilis is added as a separate undifferentiated group because of the small number of cases. The figure representing the number of Wassermann-fast congenital cases cannot be accepted as an index of the occurrence of this phenomenon in this special type of the disease, since there is a great preponderance of patients with acquired syphilis in this clinic.

Wassermann-fastness appears more often in neurosyphilitic patients than in any other type, cardiovascular and bone disease coming next in the order of frequency. Apparently well patients, classed as latent, and those with visceral disease appear to be relatively infrequent. Anatomic localization of the disease process, therefore, appears to be of etiologic significance in the determination of Wassermann-fastness. Of the patients with lesions in more than one anatomical system, all had neurosyphilis, cardiovascular, or bone disease. Combinations of neurosyphilis with cardiovascular disease and bone syphilis in congenital patients are, we think, of striking significance in their bearing on Wassermann-fastness.

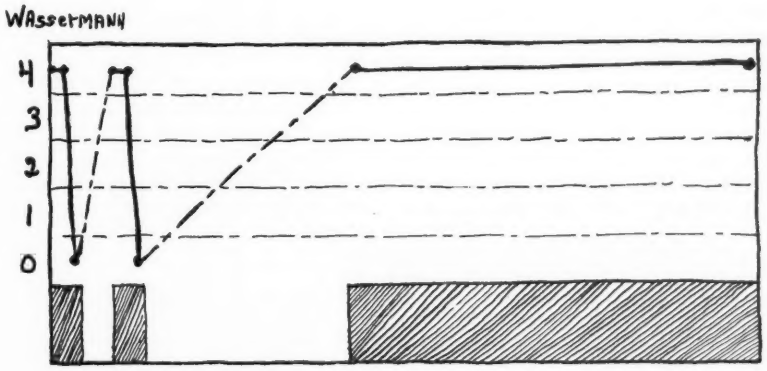
To demonstrate that the infection persists in a Wassermann-fast patient is not always easy. The most satisfactory evidence is demonstration of active lesions, as may be done in neurosyphilis with a characteristic abnormality in the spinal fluid and progressive clinical neurosyphilis; in bone lesions with typical signs and chronic relapsing course; and in recurrent lesions generally, following abandonment of treatment.

Less satisfactory evidence is afforded in the ultimate reversal of a positive Wassermann by treatment, after a period of fastness; in cases of practically incurable disease which continue to deteriorate in spite of treatment; and in patients with damaged cardiovascular systems due to syphilis, renal complexes, and more or less vague visceral disorders recognizable as syphilitic in origin, in whom the presence of active syphilitic processes cannot be either affirmed or denied by methods of study now available. It is these and a small number of asymptomatic, apparently physically fit patients who make the proper interpretation of an apparently fixed Wassermann reaction difficult if not indeed impossible of correlation with the presence and persistence of the syphilitic infection.

Studied from this angle (*Table II*) we find that conclusive proof of persistence of the in-

CHART II.

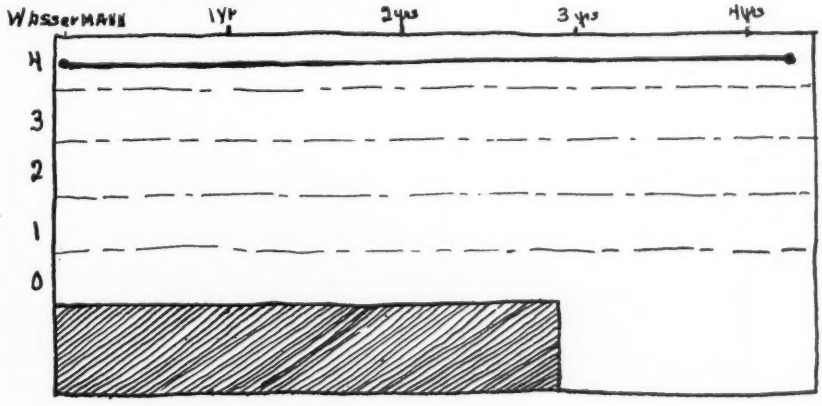
W.M. 33	Diagnosis Secondary	Treatment 14 Arsph. 0.4	Lapse 4 weeks	Diagnosis Kerato. iritis	Treatment 14 Arsph 0.4	Lapse 6 mos.	Diagnosis Hemiplegia Recurrent Secondary	Treatment 28 Arsph 0.4 6 mos. Hg+KI



Shaded = Treatment
Unshaded = Lapse

CHART III.

W.F. 38	Diagnosis Gumma Nasal Septum	Treatment 46 Arsph 0.3 10 Silver 0.2 33 Flumerin 8 mos Hg+KI	Probation 23 mos.	Diagnosis Gumma Nasal Septum	



Shaded = Treatment
Unshaded = Probation

fection in this entire group is furnished by 40 patients whose spinal fluid showed abnormalities other than the Wassermann reaction characteristic of neurosyphilis, and by 16 who developed clinical recurrence after treatment had been withdrawn. Taken together these 56 patients represent 47.5 per cent of the entire group.

Of the remaining 62 patients there were 4 with whom it was possible ultimately to reverse the previously "fast" reaction by further extension of the treatment period; 8 in whom there was further extension of the disease process in spite of continued treatment; and 46 in whom serious damage due to syphilis could be demonstrated, such as characteristic cardiovascular, or bone lesions, but in whom active syphilitic disease could not be proved beyond a doubt at the time of final study. The remaining 4 patients represent the latent group, originally 7 in number, 3 having been rendered Wassermann negative after continuation of the treatment beyond the period of one year.

Of 118 Wassermann-fast patients therefore it could be proved that 47.5 per cent were still actively syphilitic; in 49 per cent the activity of syphilis could be neither denied nor affirmed, and only 3.5 per cent were clinically well and healthy.

The following briefly summarized case histories will serve to illustrate some of the clinical

CHART IV.

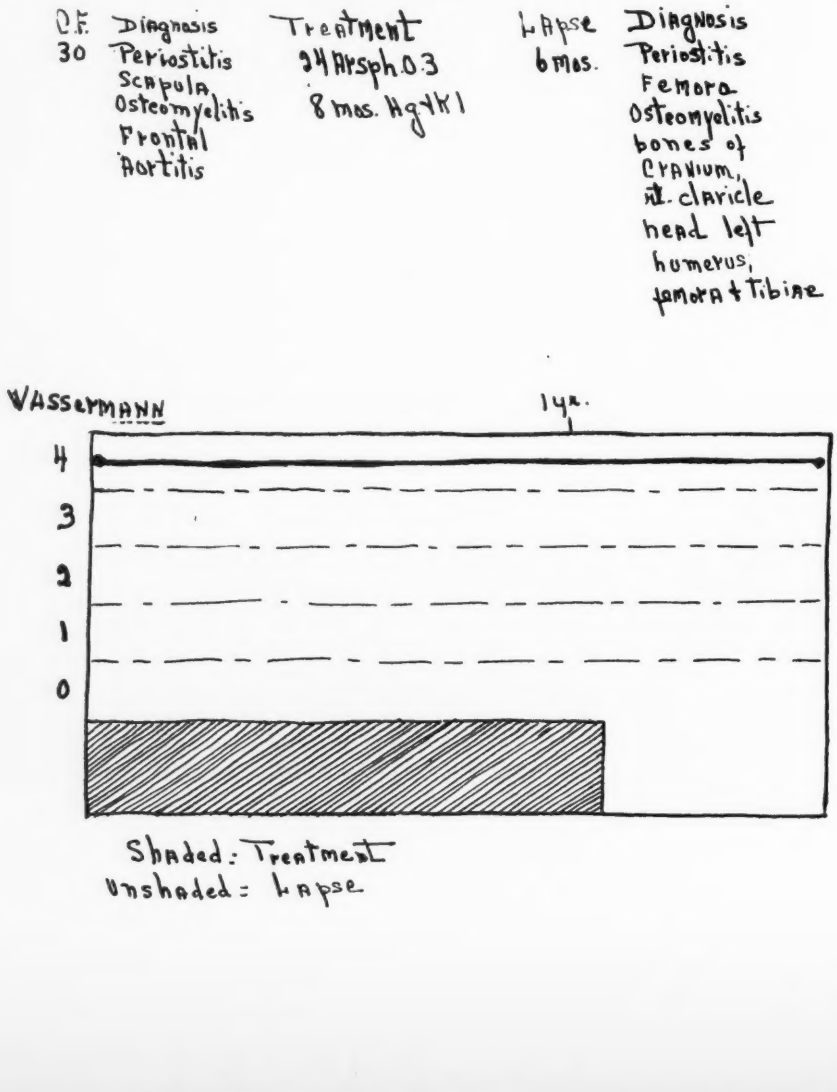
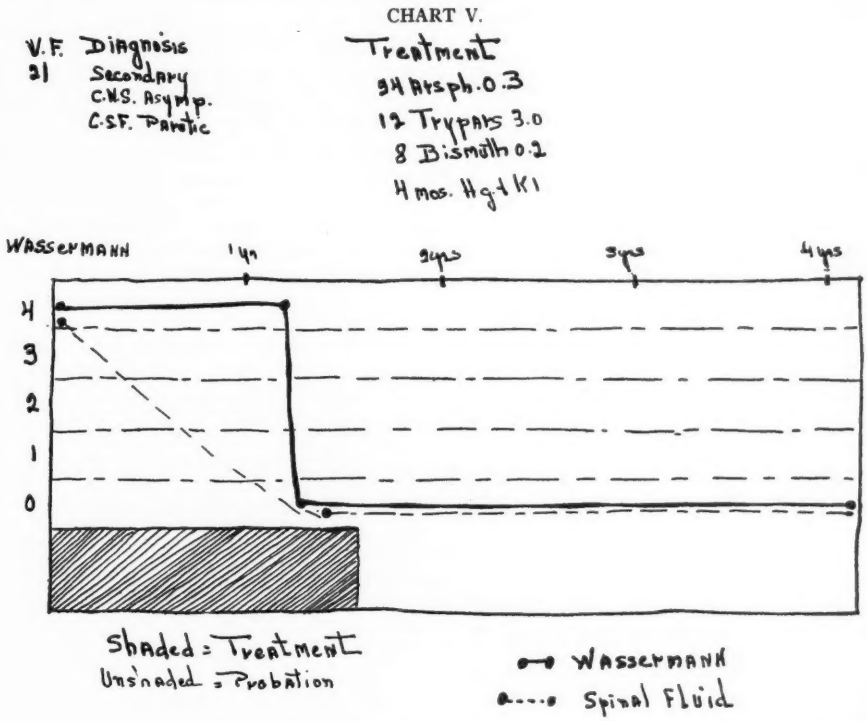


CHART V.



types of Wassermann resistant patients encountered, and the character and extent of the treatment given.

H-9683. (*Chart I*)—September 23, 1924, a colored woman, age 31, where case was diagnosed gumma of the pharynx, received 6 weekly intravenous injections of arsphenamin aa gm. 0.3; 2 months of mercury inunction, and potassium iodid by mouth. After a lapse in treatment of 6 weeks duration, she developed a perforation of the nasal septum. In the succeeding 48 weeks, she received 26 intravenous injections of arsphenamin, aa gm. 0.3; 8 intramuscular injections of bismuth salicylate aa gm. 0.2, and 4 months of mercury inunctions and potassium iodid by mouth. Her lesions healed but the blood Wassermann remained completely positive throughout.

G-60864 (*Chart II*)—May 16, 1924, a white man, age 23, was seen and his case diagnosed secondary syphilis, with papular syphilide. The duration was 6 weeks. He lapsed in treatment after 4 weekly intravenous doses of arsphenamin aa gm. 0.4. One month later he developed kerato-iritis which yielded to 4 similar injections. After another lapse of 6 months he appeared with recurrent cutaneous lesions, complete right hemiplegia and spinal fluid abnormalities of the paretic formula. Prior to this time his blood Wassermann had responded at once to treatment, but from this time on it remained a fixed positive during a year of active, continuous treatment.

G-10250 (*Chart III*)—May 15, 1920, a white woman, age 38, presented a gummatous perforation of the nasal septum. In the following 3 years she received 46 injections of arsphenamine aa. gm. 0.3, 10 injections of silver arsphenamin aa gm. 0.2, 33 injections of flumerin aa gm. 0.1, and 8 months of potassium iodid, and mercury inunctions. Her lesion had long been healed, and although her blood Wassermann was still positive treatment was abandoned February 8, 1925, she developed a fresh gummatous process in the nose.

G-59466 (*Chart IV*)—January 24, 1922, a colored woman, aged 30, was seen with periostitis of the scapula, osteomyelitis of the frontal bone, and aortitis. She received 14 months of treatment composed of 24 injections of arsphenamin aa gm. 0.3 inunctions of mercury and potassium iodid by mouth for 8 months. Six months later she returned with generalized bone syphilis.

G-73642 (*Chart V*)—November 8, 1922, secondary syphilis and asymptomatic neurosyphilis with paretic formula in the cerebrospinal fluid were observed in a white woman, age 21, who was then treated for 14 months before the blood Wassermann became negative. She had 24 injections of arsphenamin aa gm. 0.3; 12 injections of tryparsamide aa gm. 3.0; 8 injections of bismuth salicylate gm. 0.2 and mercury inunctions with potassium iodid by mouth for 4 months. Eleven months after beginning treatment her spinal fluid was less positive, and it became negative about the time her blood Wassermann was reversed.

H-11084 (*Chart VI*)—October 15, 1924, a white man, age 44, aphasic, mentally disorientated, and with bilat-

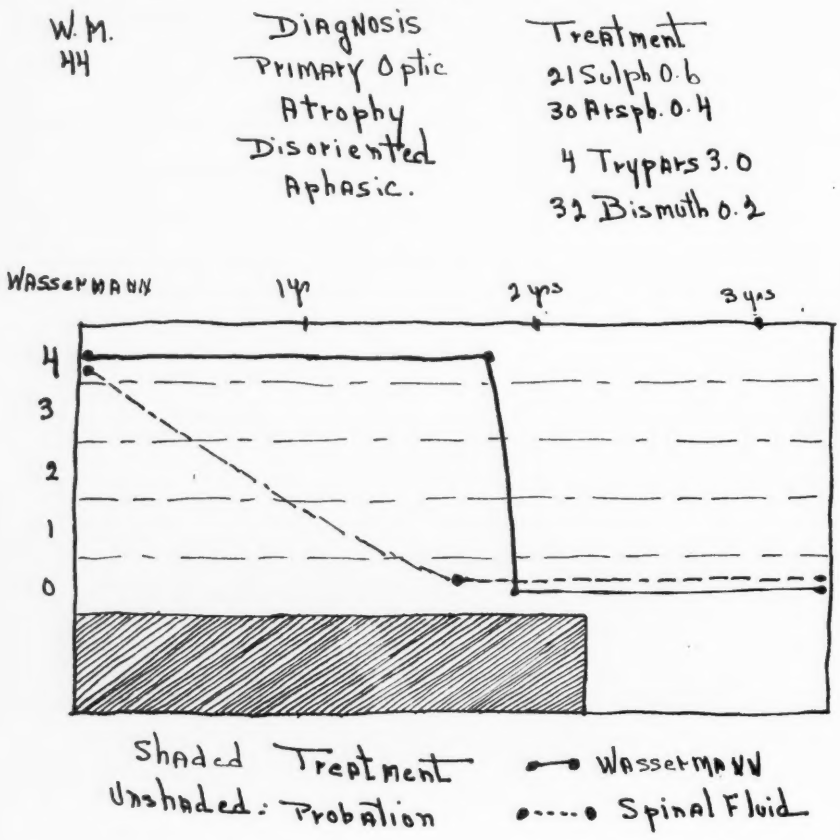
eral primary optic atrophy was given 21 injections of sulpharsphenamin aa gm. 0.6; 30 injections of arsphenamin aa gm. 0.4; 4 injections of tryparsamide aa gm. 3.0; and 32 injections of bismuth salicylate aa gm. 0.2. During the 20 months of treatment his blindness became complete but his mental status was strikingly improved. The blood Wassermann remained positive until the spinal fluid cleared up, and then 3 months later suddenly became, and has since remained negative.

On the basis of clinical evidence alone all but 7 of this group of 118 patients are known to have harbored the *Spirocheta pallida* and to have been the subjects of serious syphilitic disease. Proof of the persistence of the infection, based on the demonstration of active progressive disease processes at the analysis period is wanting in 52.5 per cent. However, other facts brought out in this study supply evidence tending to support the conception that Wassermann-fastness depends on the persistence of the infection. These facts, while not incontrovertible, lend support to the argument that Wassermann-fastness is not a phenomenon of immune reaction independent of the persistence of the infection.

It has been shown that Wassermann-fastness is present only in chronic syphilitic disease, in which, it is true, immune reactions have had full opportunity to develop. But Wassermann-fastness is confined almost exclusively to patients with central nervous system, cardiovascular and bone syphilis, both in the acquired and congenital type of the disease, and it is syphilis of just these systems which is clinically most difficult to cure.

Wasserman-fastness in central nervous system syphilis is usually dependent on the disease

CHART VI.



processes within the meningeal envelope as illustrated in cases II, V and VI, and in this sense may be a measure of the impenetrability of this envelope to drugs in the circulation, with the resultant difficulty of eradicating the infection in a special anatomical domain. Under conditions such as these it is to be expected that the blood Wassermann reaction will remain fixed until the central nervous system lesions become inactive or healed.

Wassermann-fastness has not been found in well treated early syphilis, the type of case in which the greatest probability of cure is afforded by proper treatment.

Wassermann-fastness cannot be said to have no significance because of the absence of demonstrable lesions. There are all too frequent instances of late relapses in these cases. It must be accepted as indicating a possibility of relapse at some indeterminate time in the future.

SUMMARY AND CONCLUSIONS

All patients fulfilling the criterion of Wassermann-fastness in the syphilis division of the Johns Hopkins Hospital between October, 1918 and January, 1927 were studied.

In 56, or 47.5 per cent, it was possible to prove active syphilitic disease in spite of one year or more of regular and active treatment.

In 58 more, or 49 per cent, serious damage due to the *Spirocheta pallida* was demonstrated, although the activity of the disease process could not be proven beyond a doubt.

There were only 4, or 3.5 per cent, in whom no evidence of serious damage could be found.

There were no cases of primary or uncomplicated secondary syphilis whose Wassermann remained positive at the end of one year of regular and active treatment.

We interpret these findings to signify that a fixed positive blood Wassermann reaction persisting in spite of regular and active treatment means continued activity of the syphilitic disease in the patient presenting this reaction.

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DISCUSSION (*Abstract*)

Papers of Dr. Murrell, Drs. Smith and Kimbrough, and Dr. Lord.

Dr. Jack W. Jones, Atlanta, Ga.—To the dermatologist and syphilologist supposedly well versed in syphilis, I do not think the question of a time limit in treatment will ever appeal very much; for the reason that each case of syphilis should be considered on its own merits. Some cases require more treatment than others and one cannot be dogmatic in treating the disease.

The big point which Dr. Smith's paper brought to my attention is the fact that today, when many cases are dying of heart disease, this is one cause of heart disease which can possibly be eliminated by early diagnosis and vigorous treatment.

With our present knowledge of the actual cause of the Wassermann reaction, we can only theorize and speculate upon the meaning of Wassermann-fast syphilis. The papers of Dr. Murrell and Dr. Lord are examples of the many different opinions which may be given in a consideration of Wassermann-fast syphilis. Wassermann-fast cases have seemed to me to be due to a continued activity of the syphilitic disease, probably connected with drug fastness.

Dr. C. F. Lehman, San Antonio, Texas.—All three of these papers give warning about handling the early syphilitic infection.

Dr. Smith has pointed out that the average interval between the cardiac failure and actual infection was 22 years. Of course he can not tell us how these patients were handled when they were in the early stages. We are all still hopeful that with thorough syphilitic therapy cardiac disease can be arrested and possibly cured in the future. In another two decades it will be interesting to compare such statistics as Dr. Smith has given us with the compilation of cases that have had persistent, systematic, regular treatment, over a reasonably long period of time during the early stage of their infection.

A significant point that Dr. Lord makes is that there was no case of primary or secondary syphilis with Wassermann fastness. There was no case of Wassermann-fast syphilis in primary or secondary stages, and most of his cases were chronic with involvement of the cerebrospinal system, bone or heart. Many factors combine to make treatment of the individual case a complex problem: such as the resistance of the patient, and the tecnic of treatment.

Dr. Murrell has emphasized the importance of the time factor. To my mind this has been overlooked generally and it can not be stressed too much.

Dr. Loyd Thompson, Hot Springs, Ark.—Dr. Murrell spoke about salivation which was the criterion of treatment in the early days of syphilis. There are still many men who feel that their patients should be salivated. I have seen just recently a patient who

had been treated by inunctions of mercury for a month and at the same time was given injections of bismuth. He had the worst case of salivation I ever saw, and his home physician had said to him, "Now you are cured because we have salivated you!"

The time element is important. I do not think anyone is going to turn over a patient as cured until a certain time has elapsed, regardless of the patient's condition. Everyone still clings to that. I have always been an advocate of the theory that the Wassermann test is only one symptom of syphilis, but one which means the presence of living spirochetes in the body. It seems to me that the preponderance of evidence is for that theory. I have seen only one case in which I had any doubt about this. I have of course seen many cases which showed no clinical evidence of syphilis, and if their tests were still positive, I felt that they were not cured. On the other hand we see cases, as Dr. Lord reported, in which the test is made negative by a certain amount of treatment, the patient runs along an indefinite time without any symptoms, then he develops definite symptoms of syphilis; and, at the same time, his Wassermann shows positive. These are the cases which point the way and show us that a patient who shows a positive test is a subject of scrutiny, possibly over his entire life time. He should be watched at very frequent intervals.

Dr. Davis W. Goldstein, Forst Smith, Ark.—The responsibility for the future welfare of a patient with syphilis rests with the first physician who sees the case.

Diagnosis should be made early and treatment instituted early. The patient should be under observation the rest of his life, because "Once a syphilitic, always a syphilitic."

Dr. J. Lee Kirby-Smith, Jacksonville, Fla.—The Wassermann test is valuable in verifying a clinical diagnosis of syphilis, but not to be relied upon to indicate the amount or duration of the treatment of the disease. It is my belief that if syphilis is undiagnosed and untreated for a number of years the patient will always be a syphilitic and the blood will show a positive Wassermann. If it is negative for a brief time under intensive treatment it will revert to positive. To effect a permanent cure of syphilis it is necessary that we recognize the disease in the early stages and treat the patient thoroughly and carefully, not a few months, but at intervals for at least two years, regulating during that time every feature of his life.

Dr. F. J. Eichenlaub, Washington, D. C.—I believe the crux of the Wassermann-fast situation is this: how much treatment does a positive Wassermann call for?

The cases reported by Dr. Lord were patients with active syphilis, demonstrable, clinically. This type of patient should of course, receive treatment. The patient who is mistreated is the one who has no positive clinical evidence of syphilis, has a negative spinal and who, nevertheless, just because of a positive Wassermann, is given intensive treatment. Particularly in patients past middle life, this course of treatment does more harm than good.

Dr. Earl D. Crutchfield, Galveston, Texas.—I was interested in Dr. Murrell's presentation of the side of syphilis that takes us back, perhaps a few years to the time when we cured as many syphilitics as we cure now. In the old days of mercury we cured as many syphilitics as we do now; though I think they are clinically improved much more now than they were in the old days of mercury.

There are many spontaneous cures of syphilis. I have seen syphilitic children of fathers and mothers who had no clinical evidence of the disease, and even a negative Wassermann. They lived out their lives. I think it is a biological cure.

The time factor is a very important one, but I do not know where we are going to set the time limit. One man may advocate three courses of arsphenamin; another four courses, and still another five. One man may be well in a year, another in twelve years, or not at all.

Dr. Lord's paper confirmed in my mind the view that the patients who have persistently had a positive Wassermann harbor somewhere active spirochetes. We should consider syphilis as we do pneumonia and tuberculosis and treat the patient, until he is well. But if he has a positive Wassermann, but no clinical manifestations of syphilis, it is an injustice and a great wrong to him to give him a very potent and toxic drug, which will shorten his life ten years when he might otherwise live out his expectancy.

Dr. Bedford Shelmire, Dallas, Texas.—Data on the curability of syphilis are not available. We must first formulate accurate criteria for a cure. Many cases of syphilis, undoubtedly, have been cured. I have seen unmistakable examples of re-infection in cured syphilitics.

We can follow no rule of thumb in the treatment of syphilis. A recent infection in a robust individual demands heroic, intensive treatment. The aged, with merely a positive blood Wassermann and with few or no clinical signs may not demand treatment at all. In such cases the host and the spirochete seem to live in perfect harmony. A symbiosis has been effected. Certainly the spirochete is doing the individual far less harm than would intensive mercurial and arsphenamin medication. Cases between these two extremes demand careful consideration of the therapy to be employed.

Dr. Everett S. Lain, Oklahoma City, Okla.—Wassermann-fast syphilis has been under discussion for many years. Dr. Engman of St. Louis, some years ago, was of the opinion that those cases which had been given intensive arsphenamin and mercury treatments, and still showed a positive Wassermann, might be given a period of rest, after which time, some of them would be negative.

I have tried this in several cases, and verified his opinion. I too, am in doubt as to the real dependability of a positive Wassermann to indicate the progress which our patient may be making toward complete cure. I can recall in my own experience, several cases which ran a one- or two-plus Wassermann, and were observed over a period of several years, who now, insofar as anyone can possibly determine by ordinary examination, are entirely well.

Dr. John A. Devron, New Orleans, La.—In New Orleans if cases are clinically positive we as dermatologists treat them with mild doses of arsphenamin or preferably with mixed treatment. If the patient with a positive Wassermann is between 60 and 70, before you give him arsphenamin, you should think over the matter carefully, because very often he will die of arsenical poisoning. To bring on a death of this kind is a crime.

Dr. Murrell (closing)—Are there 3,000,000 syphilitics in the United States? If there are there are at least 30,000 who come in this class of the Wassermann-fast. They are people with no demonstrable syphilis other than the serological report, after adequate treatment.

Surely they constitute a large enough class to deserve special consideration.

Dr. Smith (closing)—There are important factors in the cure of syphilis other than the use of specific drugs. The stimulation of the patients' own defensive mechanism is desirable. All of the methods spoken of as secondary measures in the treatment of syphilis should be employed.

In regard to cardiovascular syphilis and treatment there is a point which is very often forgotten, that is, that treatment of syphilis may be the cause of cardiovascular disease since the treatment may damage other body tissues. However, it is justifiable in order to cure the syphilitic disease to risk chronic nephritis later in life.

Dr. Lord (closing)—I think every patient who has a persistently positive Wassermann is actively syphilitic, the infection not having been eradicated by treatment. Some day we may evolve a treatment which will reach these processes. I do not advocate an invariable rule for the treatment of every patient who has a positive Wassermann. At the same time, if the focal lesion is in the central nervous system or in some vital organ it is imperative to treat the patient until the lesions are inactive or are healed.

Latent syphilis, by which we mean asymptomatic, Wassermann-positive syphilis, forms a large diagnostic group, second only to early syphilis, in our clinic. Yet in this group only 7 of the 118 Wassermann-fast cases were found. I wish to emphasis this point as supporting the evidence that there must be active syphilitic processes resistant to treatment on which Wassermann-fastness depends.

EPIDEMIC ENCEPHALITIS*

By CHAS. R. RAYBURN, M.D.,
Norman, Oklahoma.

Encephalitis may be simply defined as inflammation, that is, generalized inflammation, of the encephalon or brain. This paper is limited to a discussion of epidemic encephalitis. This disease is the occurrence, usually in epidemic form, of cases of subacute inflammation of the brain, due to some infective or toxic-infective agent, with a tendency toward localization in some group or groups of brain cells, especially those groups of the brain stem. In the discussion of epidemic encephalitis, there are three phases which are of more interest to us and therefore demand more attention and study than all of the others: the etiology, symptomatology and, to a lesser degree, the history of the disease. Statements of the diagnosis, treatment, pathology and prognosis, may be summarized in brief paragraphs. There is no limit to description of the sequelae. There is no limit to the vastness of the symptoms of the chronic and post-encephalitic conditions, both of which I think exist.

More time and space is given to symptoms and diagnosis in the acute and early inflammatory stages. If a few points can be brought out which will aid in the diagnosis of this disease early in its onset, this paper will have done what I most desire. For the reasons given above, this presentation is arranged with consideration first, and principally, to the acute inflammatory processes, and is closed with a brief consideration of the chronic and post-inflammatory diseased patients, with some pictures of a few of our most interesting ones.

ETIOLOGY

Before undertaking to discuss the theories and experimental work which has been done relative to the cause, it is well to state some of the known facts.

(1) The portal of entry into the brain is definitely limited to the circulatory system. The possibility of entry by way of the cranial nerves or spinal cord is very meager.

(2) The disease may occur at any time in life, some forms of intrauterine encephalitis having been reported.

(3) Results of the disease have been reported more in adults than in children. There is some evidence that it is more frequent among children. During the years of their great susceptibility to the various acute infections, it is common. The reason more cases are reported in adults probably lies in the fact that any change in the mentality of an adult would be more quickly detected than in a growing child; and the post and chronic infection syndromes of adults usually present some neurological findings, such as paralysis or myoclonias, while in children the neurological symptoms are less prominent and may be entirely absent, the most prominent symptoms being behavior disorder and a change in the moral conduct or personality.

(4) Epidemic encephalitis certainly appears to follow in the wake of the acute infections, particularly those infections which at their onset involve the respiratory tract as a whole and especially the upper respiratory tract with the nasopharyngeal region. Among the infectious diseases which frequently precede the onset are influenza, measles, whooping cough, pneumonia, diphtheria, scarlet fever, and typhoid.

(5) Trauma, although not frequently a contributing cause is certainly a factor of no small importance and must be considered. By trauma,

*Read in Section on Neurology and Psychiatry, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

I do not mean solely physical injury as from a blow, but trauma, caused by mental and physical strain such as occurs in loss of sleep, worry and overwork.

The bacteriological cause is a real problem. Drs. Neustaldter, Hala, and Bonshoff of New York, after having obtained several cases of experimental encephalitis in rabbits, came to the conclusion that the disease is caused by a specific virus which is nonfiltrable through a Berkefeld filter, and can not be cultured by any of the ordinary aerobic or anaerobic cultural methods. Many investigators have thought that some relation existed between anterior poliomyelitis and epidemic encephalitis. Much work has recently been done upon a relation which seems to exist between herpes and epidemic encephalitis. Flexner, Potts, Levaditi and others have done experimental work along these lines, and feel that some such relation exists, also Alfred Schnabel of Germany and Kling, of Sweden. The former in an article published on June 3, 1924, makes the statement that "Virus derived from cases of encephalitis corresponds closely, and may even be regarded as identical with the virus of febrile herpes." Kling, on the other hand, "considers the herpetic virus and that producing epidemic encephalitis as being different," but closely related organisms. He has isolated a virus which bears his name, and which produces lesions resembling those of encephalitis.

One great fact that should stand out in the etiology and will do so more and more until the exact cause or causes of this mysterious and difficult disease is proven, is that there has existed some relation between influenza and the onset of the disease. This is proven by the fact that the epidemics of 1768 and 1835, and the pandemic of 1918 followed the epidemics of influenza at about the same time. The symptoms of the onset and the active stages of both diseases are frequently very similar, especially when the encephalitic attack is mild.

Whether the causative agent is specific or nonspecific, whether it is due to a micro-organism, filtrable virus, or toxin formed in the brain or elsewhere in the body and reaching the brain substance because of lowered bodily resistance and dilatation of cranial blood vessels, as in influenza, remains to be solved. Such men as Charles L. Dana, Hans Zinsser, H. Hoppe, C. S. Potts, Max Meyer, and Dr. Barker have noticed the relation between the two diseases. Dr. Dana says, "there is evidence that an attack of influenza predisposes to this infection." Hans Zinsser states, "epidemics seemed to have occurred in the trail of influenza outbreaks," and "the relationship of the disease to influenza is vague, but something to be very seriously considered." Others have made similar statements. Dr. Meyer speaks of it as "grippe encephalitis."

SYMPTOMS

There are two distinct groups of symptoms. The first group is those given during the stage of onset and during the acutely active inflammatory process. The second group is those given by the chronic inflammatory and post-encephalitic conditions.

In the active stage, the initial symptoms are usually gradual in onset. There is a low fever, somnolence, drowsiness, stiffness of somatic muscles and general body aches, all of which may have been preceded by headache, general somatic pains, soreness and catarrhal states of respiratory or gastro-intestinal tracts. Headache is usually not present except in early stages. Thick speech, difficulty in swallowing, expressionless facies, and areas of paresthesia may be present. Reflexes vary throughout the course of the disease. The onset is sometimes very sudden and may simulate brain embolism, or cerebral hemorrhage, coming as a bolt from a clear sky. Delirium at the onset and in the active stage may occur. The mental symptoms may resemble some form of mental disease such as, dementia precox, mania, paresis, or infection psychosis.

Lethargy or drowsiness which was frequently present a few years ago is a state from which the patient can usually be aroused, only to go back again into the same condition. Occasionally the stupor is so deep that the patient cannot be aroused. The stuporous condition may be continuous for a short period of a few days, to months. It may be intermittent in character and last, sometimes for years. In some cases where lethargy is present as a prominent symptom, the active stage may extend over a period of several months to a few years.

Eye symptoms in some form are present in almost every case which presents enough symptoms to be noticed. They vary considerably. The most common perhaps is diplopia, which is frequently mild and transitory in nature. The next most common eye symptom is ptosis, especially of the right eye. Other eye symptoms are sluggish pupillary reflexes and, occasionally exophthalmos. Fundus changes, though rare, do occur, and may consist of optic neuritis, choked disc, hemorrhagic retinitis, and purely retinal hemorrhages.

Other neurological symptoms depend on the localization of the inflammation and may consist of: meningeal, cortical, basal, ganglional, bulbar, or cord symptoms. The neurological symptoms may be entirely absent. The shortness of duration of the active acute stage is no direct proof that the post-encephalitic condition will be absent or of meager importance. As the active acute stage subsides, and the edema, cellular exudate, and congestion is gradually relieved, there is always an abatement of the symptoms to some extent. Should the site of the lesion be away from the cerebral cortex, the neurological symptoms may apparently clear up, the same is true of some of the cases of cortical inflammation. A few of the lighter attacks which have been called abortive encephalitis never develop sequelae.

Should a patient recover from the active stage without retaining neurological or mental symptoms, and then, at some later date, begin to present post-encephalitic symptoms, these symptoms may be expected gradually to increase in severity. Between the subsidence of symptoms of the active stage and the onset of the post-encephalitic syndrome, there is frequently a very varying interval. This asymptomatic period may be days, months or years in duration. The most commonly noticed is from 1 to 3 years. Cases have been reported with the period ranging from immediately after the process to 5 years later.

The disease may have a chronic onset and attack the cerebrospinal axis without any of the general symptoms of an infection. (In this condition, the symptoms are those for chronic encephalitis except that they gradually become more prominent, and are to be looked for especially in children.) The disease at times gives symptoms of involvement of brain, cord, and peripheral nerves (neuro-myelo-encephalitis).

The laboratory findings in the acute inflammatory stages vary to some extent. There are a few fairly constant findings. The greatest aid of the laboratory, however, is that aid which it furnishes in making a differential diagnosis from many other diseases which simulate epidemic encephalitis and which have definite laboratory findings. The spinal fluid examination is one that should be done. It is a procedure which acts as a check on any case having a doubtful diagnosis of encephalitis. The findings depend to some extent upon whether or not the meninges are involved, and if the seat of the lesion is well away from them, the spinal fluid is likely to be negative. In some of these cases, it is possible to find a pressure which suggests some increase above the normal. The fluid is clear. A slight degree of turbidity has been reported. Globulin and cell count, if abnormal, are increased, and their amount of increase depends directly upon the amount of meningeal irritation. Robert Kilduff in the *American Journal and Record* reports his findings on pleocytosis to range from 2 to 212. Sugar content is usually increased and has an average of about 0.0588 per cent whereas the normal is about 0.0528 per cent. Complement-fixation tests are negative and bacteriological examination yields negative results. The colloidal gold curve gives no typical reaction and varies from negative to either a typical luetic or even a typical paretic curve. In the post-encephalitic conditions, the spinal fluid usually yields negative results.

Blood examination in the active or acute stage usually shows a slight leucocytosis. In general the maximum total white count is around 12,000. The more severe the symptoms, and especially the more sudden the onset, the greater is the leucocytosis. Over a period of some six weeks, in one patient with fairly severe symptoms of the active process, I made several blood counts and the total count ranged from 12,000 to 13,000 consistently. It is possible, that in some cases, the count could be found somewhat higher. It is more likely, however, to be from 8,000 to 12,000, with 70 to 80 per cent polymorphonuclears. In the chronic or post-inflammatory stage, there is frequently lymphocytosis of a mild degree, instead of a leucocytosis, ranging from this down to a leucopenic state. All investigators report that blood cultures give negative results.

In taking up the symptoms of the chronic and of the post-inflammatory conditions, it must be remembered, that there is frequently a very varying interval of absolute absence of all symptoms; that the chronic encephalitic condition which is to progress, may appear just before, during, or immediately following the onset. It may appear days, months or years subsequent to the active process; and these symptoms have a direct relation, and depend almost entirely on the site of the lesion. Again, the manner in which the site of the active process remains as the inflammation subsides, is to be recalled. First, the inflammatory process, if mild, may leave and no scar tissue or further sign of its one time presence may remain; second, a scar of neuroglia tissue may remain at the site of the former inflammatory process and produce a constant symptom or group of symptoms; and third,

in the scar of neuroglia tissue a cyst may begin to form (which is very characteristic of scar tissue of the brain) and gradually increasing in size, produce increasing neurological and mental symptoms. It is probable that several of those cases in which there is an asymptomatic period for months or years, after the subsidence of the active stage, and which then begin to show symptoms of a gradually increasing severity, are of this third type.

There are no two patients who present exactly the same group of symptoms either in chronic encephalitis or in the post-inflammatory types. There are so many types of these two conditions that I am giving a simple classification into which nearly all patients presenting these syndromes may be divided.

Among the mental and psychic phenomena which frequently occur are the following:

(1) Behavior Disorders
  (1) Irritableness
  (2) Laziness
  (3) Suspiciousness
  (4) Lack of concentration
  (5) Tendency to annoy and torture weaker individuals
  (6) Untruthfulness
  (7) Disturbance of temperament
  (8) Absence of any regret for behavior
  (9) Kleptomania

(2) Personality Changes of
  (1) Limited emotional responses
  (2) Emotional instability
  (3) Insufficient mental energy
  (4) Dull complaining monotony
  (5) Ambition at low ebb
  (6) Catalepsy

Among the neurological conditions which may be found, the following are probably the most frequent:

(1) Parkinsonian (paralysis agitans) syndrome
(2) Chorea-athetoid movements, bodily contortions, Paroxysmal "stretching," and various myoclonic twitchings.
(3) Salivation
(4) Spastic paralyses
(5) Epilepsy
(6) Respiratory disturbances and cyanosis
  (1) Disorders of rate of respiration
  (2) Disorders of respiratory rhythm
  (3) Respiratory tics
  (4) Any combination of these three
(7) Sleep disturbances
(8) Hiccough
(9) Trophic disturbances
(10) Vasomotor disturbances
(11) Temperature disturbances
(12) Areas of anesthesia
(13) "Shooting pains"
(14) Any admixture of these

The course of the disease varies, both in the active inflammatory stage and in the post-encephalitic syndromes. There is a tendency to remission. Some patients may apparently have recovered or be recovering when involvement of some vital center, as the cardiac or respiratory, will result in sudden death. The mortality varies from 10 to 25 per cent. In some cases, the disease may be so mild that it is thought to be only a cold or even a light attack of influenza. Patients sometimes do not even discontinue their routine of daily duties.

Most encephalitic conditions are chronic and progressively deteriorating. Some few patients improve. A few cases of recovery have been reported. The duration extends over long periods of from a few to several years, depending on age, environment and responsibilities of the patient.

PATHOLOGY

Any group or groups of cell bodies in the brain may be affected. The process affects primarily the gray matter or cell bodies, but secondary involvement and degeneration of white matter or nerve fibers is frequent. The most commonly affected areas are the nuclei of the corpus striatum, that is, the globus pallidus, the putamen and the caudate nucleus. Other areas commonly involved are the hypothalamus, substantia nigra, red nucleus, nuclei pontis, the thalamus, motor and sensory cerebral cortices, the higher respiratory centers, and different psychical areas. One cause of the frequent involvement of the corpus striatum and the subcortical regions undoubtedly lies in the nature of their blood supply. Both of these areas have terminal arteries.

Regardless of the region of the brain in which the lesion is located the microscopic picture presents more or less similar characteristics in all. This, in the active inflammatory stage, consists of:

(1) Distention of blood vessels with regional conjestion.
(2) Surrounding pia conjested.
(3) Abundant foci of punctate hemorrhages.
(4) Perivascular (especially perivenous) round cell infiltration.
  (This is the differential point between encephalitic and degenerative inflammation.)
(5) Increase of neuroglia tissue.
(6) Neurono-phagocytosis.
(7) Stages of tissue disintegration and absorption.

Subsequent to the active process, anyone of

three results may occur. First, if the inflammation has been mild, all signs of its one time presence, may leave entirely and never recur to present chronic or post-encephalitic symptoms. Second, the inflammation may subside and leave only a scar of neuroglial tissue. Third, scar tissue, which remains after inflammation subsides, may be the seat of cyst formation. This cyst may have its onset weeks, months, or years after the active process has subsided, and gradually increasing in size, produce very prominent symptoms of brain tumor which gradually increase in severity.

HISTORY

Epidemics of encephalitis resembling the disease now variously known as epidemic, lethargic, and less commonly as, hemorrhagic, polio, and hemorrhagic-polio-encephalitis, and by some, even, sleeping sickness have been described at different times during the past 450 years. "Definite descriptions of epidemics may be obtained since 1695.[1]" The present epidemic in the United States began during the latter part of the year 1918. It might here be added that the influenza epidemic swept westward over the United States during the late summer, fall and early winter of 1918. The same association existed in regard to the epidemics of 1768 and 1835. These facts are ones that anyone believing that no relation exists between influenza and our recent epidemic of encephalitis must explain.

Lethargy is a term that was probably first applied when it was spoken of as "lethargic fever, with disseminated eye signs" in describing the epidemic of 1695. The name was again applied in describing the epidemic occurring in England in 1916. There are many cases in which lethargy does not exist. Many authors, for this reason, and properly so, prefer not to use this name. While the terms, lethargic and epidemic merely describe sometimes prominent, and very frequent points, the other terms describe some of the prominent pathologic pictures occurring in the course of the disease. Since it may affect the brain, the cord, or the peripheral nerves, Potts[1] recently suggested the term, "encephalo-myelo-neuritis." Certainly some name should be agreed upon, and possibly the name of epidemic encephalitis, which is apparently fast gaining in favor and which sufficiently differentiates it from others, such as, suppurative, chemical, traumatic, syphilitic, tuberculous, and disseminated sclerosis, is the most proper.

"European epidemics of this disease are known to have occurred in 1712, 1718, 1745, 1768, 1800, 1802, and 1835. In 1889 and 1890, it appeared in Italy, Hungaria, Germany, and France." In 1892, under the term of "nona," it was well described by Longuet. In about 1916, a pandemic started in Austria and Australia, occurred somewhat later in England and France, and began in the United States during the latter part of 1918. "It is peculiar, that whenever it has appeared, it has been thought to be a new disease, and that there is a relation between its appearance and epidemics of influenza."

TREATMENT

To summarize the treatment, in the active stage, rest in bed, quiet (drug sedatives if required), warm baths, warm packs, and treatment with spinal drainage and intravenous medication of various hypertonic salt solutions or 100 per cent glucose solution as indicated for relief of intracranial pressure should be given. At present, it seems that intravenous treatment with sodium salicylate (5 c. c. of 20 per cent daily) or intravenous neutral acriflavin (10 to 20 c. c. of 0.5 per cent daily) should be tried. In the post-encephalitic conditions general hygienic measures with systematized massage, hydrotherapy, passive motion, and the use of hyoscine hydrobromid (1/150 to 1/50 grain 4 times daily) should be tried. In the severe tremors, if bulbo-capnine is available, it may be used to advantage. Most individuals with chronic encephalitis or those presenting post-encephalitic syndromes, at some time, have to be institutionalized.

Discussion follows paper of Dr. Alford, page 653.

1. Potts, C. S.: Epidemic Encephalitis. Amer. Jour. Electrother. and Radio. pp. 311-315, 1924.

THE USE OF GLUCOSE IN THE TREATMENT OF EPIDEMIC ENCEPHALITIS*

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It was quite by accident that I first came upon this procedure. Drs. Winter and Clithero called me in consultation to see a patient who was in a delirious condition. She had previously had two blood transfusions, without any noticeable effect, and the question was naturally what more could be done for her. Since she

*Read in Section on Neurology and Psychiatry, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

was refusing food it was suggested that fluids and nourishment be given in some form. These physicians resorted to glucose by vein, which they administered in large amounts. One day she received four injections of 500 c. c. of 5 per cent solution of glucose, and it was continued for about ten days, or until she began to accept nourishment by mouth. The condition ran a very severe course and for a week or more exitus was almost hourly expected; but to everyone's surprise the patient began to improve and on Christmas day, 1926, which was about a month after she was first taken to the hospital she regained her senses and on New Year's day she went home. She has remained perfectly well ever since.

This was regarded by the attending physicians as a case of encephalitis and it may be added that the course of the illness was a surprise to all. In casting about in our minds for the reason for this result it seemed possible that the free use of glucose was the responsible factor. It was decided that at the next opportunity glucose would again be tried.

The opportunity presented itself as the first patient was convalescing. This case was an appropriate one for experiment because the condition had existed and run a very even course for about four months. The patient was mildly delirious, much emaciated, had abdominal cramps and persistent diarrhea, and had developed a marked facial hirsute condition. There had been, it may be said, numerous consultations and special examinations and about everything that could be thought of had been tried out on her.

After glucose injections were started there was quite rapid and definite improvement and the patient left the hospital in about three weeks. This second experience fairly started us on our course in the use of glucose in encephalitis.

Later it was found that our employment of glucose in this connection was not entirely original. A physician of St. Louis, Dr. Fagley of the Veterans' Bureau, had been using it regularly on post-encephalitic conditions, for more than a year. When consulted about its use he stated that he first learned of it from a physician connected with Columbia University, New York City, who had personally reported good results to him. I read a copy of the letter from this physician in which he spoke highly of the effects of glucose. Dr. Fagley seemed mildly enthusiastic about his results, but it must be admitted that since, as I understand it, he was working chiefly with Parkinsonian cases, he had the least favorable type with which to deal. I have since found that others have used it to some extent and I have been told by physicians who treated large numbers of influenza cases during the war, that glucose injections were freely used in such cases. However, as far as I have been able to find, nothing has been published specifically about the use of glucose in encephalitis.

My cases are forty in number, of which twenty-two were relieved of practically all symptoms, twelve were relieved to a greater or less extent, and six were unimproved. For purposes of convenience of description the cases have been placed in groups according to the preponderating symptoms. There were first eight delirious cases, five of which were almost completely and three partially, relieved. The delirium in all but one of these cases was of short duration. The next group, nine in number, were instances of acute exacerbations of nervous symptoms which were thought to be similar to the foregoing group but of less severity. All but one of these recovered. Then there were seven cases which were designated subacute because the symptoms had existed from one to two years; of these four recovered and three were unaffected. There were five Parkinson cases of which only one can be regarded as recovered, three as improved and one as unimproved. I shall refer to these cases further in a moment. Of behavior disorders in younger persons there were four (one being a duplicate which also presented Parkinson symptoms). Two of these showed improvement in the behavior disturbance, one as to general symptoms, such as headaches and other pains, and one (duplicate) was unimproved. Three patients had convulsions and all apparently recovered completely. There was one case of marked respiratory disturbance which recovered. In a miscellaneous group of three that will be referred to again, two improved.

Where possible, glucose was administered by vein in the ordinary manner, but when this was not possible it was given by mouth, and also, in most cases, while injections were being given, administration by mouth was pursued at the same time. Although sometimes quite successful, mouth administration seemed much less dependable, slower in producing results and more apt to be followed by relapses. Of the twenty-two successful cases, seventeen received injections. Of the thirteen in which improvement was apparent, seven received injections, and of the six failures, all received injections. The figures do not seem to jibe with the above conclusions. Nevertheless better results would have been expected in a number of the oral cases if they had been treated by injections. It must be admitted that this impression has been obtained partly from cases not included in the series because treatment was not continued long enough to be considered to have had a fair trial.

There have been remissions in four of those patients in whom symptoms were at first completely relieved and in four who were improved

(five of the former and four of the latter have been lost sight of). One of those who were regarded as completely successful had a remission and is again being treated by injections with improvement. Another, the instance of respiratory disorder, has recently had a remission according to the report of the physician but I have not been asked to see her. Two, who received glucose chiefly by mouth, had quite severe remissions, but are again in a satisfactory condition.

Taking up individual cases more in detail, a few points of especial interest will be referred to.

One delirious patient who was seen by another neurologist as well as by me, was in a very precarious condition. She was a potential diabetic and glucose was given only by mouth, the urine being carefully watched. She recovered completely in about three weeks and has remained well ever since, for a period of four months.

The case of respiratory disturbance was especially interesting. The patient was intensely cyanotic, the pulse being 140 and respiration from 20 to 40, the temperature occasionally rising as high as 101.° Even after taking these factors into consideration it was impossible to account for the cyanosis which was more marked than I have ever seen it before. As there was pain in the abdomen, abdominal hemorrhage was suspected. A blood transfusion was given without effect. An order was given for 150 c. c. of 10 per cent glucose to be administered with care but the interne gave 500 c. c. instead. The patient improved very rapidly. Three more similar injections were given, and just a week after the first injection she and her relatives insisted on her going home. Her condition remained satisfactory until just recently, a period of eight months, when she had a recurrence.

In the Parkinson case, the patient had intense headaches, a good deal of weakness, general and especially in the legs, some somnolence and mild rigidity. All of the symptoms practically disappeared, and one unacquainted with the case would not now guess that a Parkinson syndrome had been present. This change was confirmed by other workers around the clinic.

Turning to the cases in which there was improvement but not complete relief from symptoms, one was a case of delirium that developed after operation, and while the delirium disappeared, there remained pains, and weakness. The physician in charge stopped at seven injections instead of giving the number advised. Another delirious patient had only five injections, and upon recovering her senses refused to take any more. Still another delirious patient had also a great deal of vertigo with retropulsion. The vertigo had preceded the delirium in onset. During the course of treatment the delirium cleared but the cerebellar symptoms persisted without much change. The patient was a woman, 66 years old, and there was some doubt about the diagnosis. At the end of the treatment she left the hospital against advice and was not observed further. By the way, several of these cases were treated almost against my desire, because, for one reason or another they did not appear especially promising, but on the chance that it might help, glucose was given.

One of the Parkinsonian patients lives in a distant city and was never seen by me. She had been ill and very miserable for two years and had been given a hopeless prognosis by her physician. She took glucose only by mouth. After four months it was requested that she, and independently her sister, should write concerning any change that might have taken place in her condition. The patient writes: "I am better in every way than I was before I took the glucose," and her sister states: "She is incomparably better than when she left St. Louis in March." But from other statements in the letters I judge that she still has many active symptoms. Another Parkinsonian patient was semidelirious, much emaciated and very weak. After a course of glucose she was very much improved in respect to the active symptoms; but there was no change in the Parkinsonian picture.

The other patient who had a very marked rigidity that had persisted for four or five years, had very few active symptoms, those present being weakness and sweating. There was no improvement in the rigidity. I have sometimes wished that I could continue injections in these cases for a much longer period, just to see if anything could be accomplished, but for one reason or another this has never been possible.

Of the three behavior disorders, one was in a girl of six who could not be controlled, was hyperactive, disobedient, and always fighting with little boys. The woman who brought her to the clinic repeatedly stated that she was much improved. Treatment was continued only for about a month.

A boy of six was a patient at the Children's Center and was followed very closely by social workers by visits to the home and school over a period of ten months. He improved to such an extent that both the teacher and the parents considered him normal, and the latter discontinued glucose of their own volition. At a recent visit however, the patient displayed some of his old symptoms and was evidently not so well off as had been supposed.

Another was a boy of twenty who had influenza nine years previously but had shown symptoms for about three years, certain behavior aspects which consisted chiefly in annoying women by calling them over the telephone and persistently writing them letters. He also had headaches, pains in the back and legs, shortness of breath and weakness. Glucose injections were given by his physician who stated that all active symptoms had disappeared, but there was no improvement in behavior.

A patient of 42 had a mild apoplectic stroke, for which no cause appeared, remained aphasic and mentally confused for many weeks. Long after the original attack he began to show great difficulty in breathing at night and to have occasional febrile attacks. It was thought that the condition had never been understood. Glucose was given by mouth on the chance that it might help. In two weeks the patient's wife stated that the acute symptoms had disappeared and the aphasia was certainly much less than it had ever been since the original attack.

Another patient was quite tremulous and weak, had fainting attacks and transitory focal weakness of one arm. Under glucose by mouth she firmly maintained that she was in every way improved, carried on her work as a canvasser without interruption for many weeks and did seem to be much better. Unfortunately, she was in the midst of the recent tornado, and thereafter symptoms returned with increased severity. She was sent to the hospital and ten injections of glucose were administered, from which there was no benefit

whatever. It is doubtful that this case should really be classed as one of improvement.

The last case in this group is that of an Irish policeman 40 years old. The complaint was weakness, fluttering of the heart, diarrhea, and the loss of 30 to 40 pounds in weight. There were spells of weakness in which he feared he would pass away, burning of the soles of the feet, and numbness of the tips of the fingers, and also periods of drowsiness. He had been ill for over two years, had had numerous tests including gastro-intestinal x-ray examination, by the best physicians in the city and all treatment had been without effect. Almost all symptoms improved rapidly under glucose by mouth, but there remained flatulence and irregularity of bowel movement, of which he complained very bitterly. He was urged to take intravenous injections, but refused, and finally disappeared from view in this condition.

Now taking up the cases which were considered complete failures, I shall mention first one of deep somnolence, the only one of true lethargy in the series. It is true the case was a severe one, but improvement was confidently expected. Nevertheless there was very little change in the condition after twenty injections and death ultimately resulted.

Another was a case of general weakness of about seven years duration, which had been seen by several neurologists, none of whom had been willing to venture a diagnosis. There was no improvement after five injections and the patient did not care to take any more. I myself am not positive of a diagnosis in this case.

Another was a case in which the predominant symptom was a severe headache. This symptom was not of long duration and when treatment was instituted we were all quite hopeful of the result; but after twelve injections no improvement was noticed and the patient passed from observation.

Another case was one of headaches with transient periods of delirium, the symptoms being of two years or more duration. After five injections the delirious symptoms were no longer noted but the headache had not improved. The patient refused to take any more injections and I have since heard that she is now in an asylum.

The next case was a bitter disappointment. The patient was seen last June and the symptoms dated from December, 1926. These consisted of severe headaches, a generalized tremor, some vertigo, spells of weakness and palpitation. It was felt that there was little doubt of the diagnosis, and that as the symptoms were all of an acute nature and of short duration improvement should in all probability follow treatment. After twelve injections of glucose there was not the slightest change of condition and I have heard just recently that it is rapidly progressing.

The last case, a male 20 years old presented a combination of behavior disturbance and moderate Parkinsonism without any acute symptoms to speak of. After seven injections of glucose there was no improvement in any respect.

Referring to the matter of diagnosis, as well as of results in these cases, you will have to accept a good deal on faith. It may be said that practically all of them were consultations and were therefore observed by other physicians, some of whom are among the leading practitioners of St. Louis. Most of the patients had all of the customary tests and examinations. Still in a certain number, as has been mentioned, there remains a question of diagnosis. As you all know, the diagnosis of encephalitis is often largely a matter of exclusion; and differentiation from certain conditions, such as mild psychoses for instance, may be very difficult.

I have come to believe that many obscure cases which one is called to examine nowadays really belong in the encephalitic group. The symptoms are indefinite but persistent and disabling. They include headache, dizziness, general weakness, loss of weight, intestinal disturbances, numbness of the hands and feet, chilly sensations, palpitation and attacks of weakness, often without noteworthy somnolence or fever. If these symptoms are really of the nature suggested, encephalitis is a far more prevalent condition than we now suppose. Furthermore considering these symptoms one might conclude that encephalitis is not merely an infection of the brain, but a general intoxication.

Almost every physician who has seen these cases has asked what is the rationale of the procedure. It is, of course, purely empirical. Some who have used glucose injections have done so on the theory that there is a withdrawal of fluid from the brain. However, I have a different idea. A 10 per cent glucose solution is not, a chemist tells me, very hypertonic, and a 5 per cent solution not so at all. Furthermore, administration by mouth should not have a hypertonic action. Some years ago, with Dr. Opie, I was engaged in some research work on the effect of diet on the action of certain liver and kidney toxins. It was found in the course of our experiments that carbohydrates exerted a markedly protective action, proteins were intermediate, while fats enhanced the toxic effect. It appears to me possible that glucose may have a protective effect on the nervous system just as in those experiments carbohydrates had for the liver and kidneys.

The usual method of administration by vein consisted of the use of 10 per cent solution, in amounts beginning at 150 c. c. and running up to 500 c. c. The injections were given daily, or as nearly so as was possible, and if it was feasible, 15 injections were given. In oral administration one to two ounces were given three times a day. Oral administration always accompanied the intravenous method and was continued for an indefinite time.

If the solution was right no bad effects were

observed even in the most desperate cases. It is my belief that if the solution is in every respect up to standard there will be no reaction. However, it must be strictly fresh. If kept for a day or two, or resterilized toxic by-products, perhaps acids, are formed and severe reactions are obtained. I am sure that acid formation is usually the cause of reaction, but sometimes, apparently, there is something wrong with the water still which permits foreign substances to enter the water. Reactions may be severe and in one of my cases, I feel certain the reaction hastened the end.

In considering the cases cited one cannot escape certain mental reservations. In the first place some cases that appeared to be hopeful showed no improvement. Again when the course was favorable one could not always be sure that this issue was entirely due to the procedure, such is the variability of the disease. Some cases no doubt improve spontaneously. Once more it must be admitted that a large proportion of the cases of the present series were of the more acute and milder types. Nevertheless after taking all these facts into consideration, it is felt that in many instances the results were sufficiently pronounced to give the procedure a definite place in the treatment of encephalitis.

By way of summary it may be stated that in general results were best in the more acute cases and least favorable in Parkinson and other syndromes where actual destruction of tissue can be assumed to have occurred.

Beaumont Building.

DISCUSSION (*Abstract*)

Papers of Dr. Rayburn and Dr. Alford.

Dr. W. E. Gardner, Louisville, Ky.—Encephalitis especially in the acute stages, presents a variety of clinical manifestations. It is not always easy to make a diagnosis of encephalitis lethargica in the acute stage. We are very likely to mistake some of the toxic exhaustive psychoses for encephalitis. We see a type of lethargy and confusion following toxic exhaustive states, which are not infrequently temporary, and there is a tendency to overlook some of these confusional states, if we are looking for lethargic encephalitis. We should bear in mind some of the outstanding characteristics of the acute stage, and while all cases do not present the typical triad of headache, fever and cranial nerve involvement, we usually have at least two of these prominent symptoms present. Almost invariably there is some cranial nerve involvement because of the location of the inflammatory process about the basal ganglia and the aqueduct of Sylvius. Especially do we have involvement of the third, fifth and sixth cranial nerves, and sometimes the fourth and seventh. I think this is important. After we have diagnosed a case as acute encephalitis considerable time may pass before we know whether or not the diagnosis has been correct. We have all seen cases with a characteristic Parkinson syndrome, giving a history of having had an attack of what was called influenza, some two or three years before, from which the patient made an apparently good recovery. There had been no intervening symptoms, and then after this prolonged period we have the development of tremor and other Parkinsonian manifestations. Examination of the spinal fluid in acute cases is important and helps to rule out other lesions of the central nervous system, but it does not rule out poliomyelitis, especially in young subjects. The spinal fluid findings in the early stages of these two diseases are essentially the same, showing an increased cell count, and increase of globulin, and an elevated sugar content. I have never been able to see the so-called typical paretic colloidal gold curve very successfully demonstrated in encephalitis, although it is said to be quite characteristic in this disease, even in the absence of any luetic involvement. The treatment of the Parkinsonian syndrome is one of the most difficult things we have to contend with, but the use of hyoscin seems to have met with a good deal of favor. Tincture of belladonna in large doses has been used in a fairly large number of cases with some success, and has been recommended for the same reason as the use of hyoscin. The use of occupational therapy in these cases is important, and should not be overlooked.

I cannot see how glucose would have a tendency to any specific action in this disease. If it does good, I believe it is in the general support of the patient in a nutritive way. It would have a tendency to bring up the alkali reserve of the blood, as it has been demonstrated that in other infective and exhaustive states there is a loss of the alkali reserve, which is perhaps an important contributing factor to the mental confusion and motor restlessness that we frequently see in such cases.

Dr. C. C. Turner, Memphis, Tenn.—Why in children, particularly young children, does epidemic encephalitis seem prone to have more of a cortical involvement than in the adult type where the basal ganglia and the hypothalamic nuclei seem to be more involved? During the epidemic in 1920 in most of our children patients, particularly the young children with encephalitis, we had a marked pia-arachnoiditis which frequently occluded the foramina of exit of the fourth ventricle, producing an internal hydrocephalus.

In referring to the treatment of this disease, Dr. Rayburn made no mention of the use of autogenous serum in cases, particularly of the Parkinson type. This treatment was recommended some few years ago. We tried it out in a few cases here and thought that there was some improvement in them.

Dr. H. M. Austin, Laredo, Texas.—In about five cases of encephalitis lethargica, I have had very beneficial effects from intravenous injections of mercurochrome.

Dr. Rayburn (closing).—Many things have been used in the treatment of these cases, which fact alone will suffice to show that there is no one thing that we can consider a definite cure or even worth very much. You have to keep one thing in mind, that is, that there has been a definite destruction of brain tissue.

I have not used mercurochrome in the acute cases. I have used acriflavin, sodium salicylate, and urotropin. Acriflavin is one of our most penetrating dyes.

So far as autogenous serum is concerned, I think if you could give the proper amount of serum in the early stages of the acute condition, you might help to stop the process, but in restoring the destroyed brain cells, I cannot see how it would be of any benefit.

STUDIES WITH UNDERWEIGHT CHILDREN IN THE FAYETTE COUNTY (KENTUCKY) SCHOOLS*

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In presenting a preliminary account of studies with a group of underweight school children we are but corroborating some of the findings of others and adding little to the present knowledge of this subject. Our interest has not been to these ends, but rather in an investigation of causes for this condition and to just what extent our department should be concerned in dealing with them.

This investigation was prompted not only because of the large number of underweight school children found, but also because of the variation in numbers which was found to exist in the county schools. To be more specific, the percentages of these children ranged from 26 to 57. The number of children with physical defects ordinarily associated with weight deficiency did not vary in the several schools in accordance with the number of underweight children. Likewise, there was no parallel between observance of health habits and underweight.

Further search lead us into other fields. For instance, a low economic status brought about by poor financial returns from the preceding year's tobacco crop in a rural district was followed by a high per cent of underweight school children. Thus straightened circumstances no doubt made for privation of food and rest to the extent that underweight predominated in this group of children.

Then again, there are some children who "come naturally" by their failure to measure up to normal weight requirements and, in the absence of acquired causes, we must conclude that their normal falls below the average standards.

We must not lose sight of such conditions as malnutrition, and endocrin disturbances, which must be left for the care of the skilled physician. There are left, then, certain physical defects and faulty health habits as important causes in maintaining a common level of underweight school children.

It so happens that the four largest schools in the county represent a fair cross section of its white population. Two of these schools draw from suburban districts, the third from a section in part suburban and in part rural, while the fourth is attended by children from a rural community. This study has been concerned with children from six through fourteen years of age who attend these four schools. The combined enrollment of these age groups was 857. Of this number 317, or 37 per cent, were found to be 7 per cent, or more underweight. These children comprised our group for study.

It is interesting to note that the social and economic levels of these four districts were found to have a direct relation to the numbers of underweight children. Location and numbers of underweights did not bear any consistent relationship, except that the rural members showed conspicuously more underweights. Studies of other districts have lead us to believe, however, that rural children are no more prone to weight deficiency than others where social and economic conditions are in common.

Some rather marked differences have been noted in the number of underweight children found at the opening of school in the fall and at the mid-session in winter. Freedom from a routine common to the school period, increased out door activity during the summer months, neglect of health habits in many cases are, as we know, important factors which reflect in little or no weight progress as revealed when these children return to school in the fall.

When this seasonal variation was determined for age groups, the greatest per cent of underweights was found among both sexes at thirteen years. This corresponds to a sensitive period of physical transition and development during which the greatest demands for weight increase normally occur. The predominance of underweights at thirteen years was most striking when age groups of boys and girls were determined at the time of the mid-school session. More underweight girls than boys were found in all age groups, except at six and nine years.

Leaving these general considerations at this juncture, it will be recalled that our underweight group was comprised of 317 children from six through fourteen years of age. At the end of a three months period we were able to select records of 290 of these children which were complete enough to enable a detailed study concerning weight trends and the relation to these of defects and routine practice of certain health habits. Some of our findings will now be considered.

Weight progress during this period was poor

*Read in Section on Public Health, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

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for the whole group, the average gain per child being but 0.5 pound per month. The group contained, however, a large majority with physical handicaps to weight increase and many with poor records of health habit practices. In fact, all but seven of these 290 children were found with some deterrent record at the end of three months. An average gain of 1.4 pounds per month for these seven children as against 0.5 pounds average for the whole group seems to substantiate the above statements.

Girls gained more than boys in every age group, the averages per month being 0.7 pounds for girls and 0.33 pounds for boys. In making a division of these groups into those from 7 to 10 per cent and 10 or more per cent underweight, we found that more than twice as many of both sexes were in the latter, and that girls were in greater proportion than boys in both.

In the investigation of physical defects and their relation to underweight, it was found that those of tonsils, teeth, vision and lungs merited consideration.

These defects were more commonly found among underweights than among others in the schools.

When curves were plotted for all children with tonsil defects and for underweights with tonsil defects, it was found that a curve superimposed for per cents of underweights in age groups bore a close relation to that for underweights with defective tonsils between nine and twelve years. At thirteen both of these curves ascend and at fourteen both decline. When average weight gains for underweight groups with and without defective tonsils are compared, all except those of eight and thirteen years made better gains in the latter group. All underweight children without defective tonsils made an average gain of 1.72 pounds per month as against 1.45 pounds for those with this defect.

Although children with defective teeth were found in greater numbers among underweights, curves plotted as for tonsil defects failed to parallel at any place. Average weight gains were 1.94 pounds for those without defective teeth and 1.53 pounds for those with this defect.

Here, as in the case of tonsils, we do not feel free to conclude that because weight differences were in favor of the group without defective teeth, the defect was solely responsible. Frankly, we were surprised to find that so great a difference as this would result where the incidence of systemic effect is relatively low. We have no school dentist to whom these children might have been referred for a more detailed examination and opinion. In the absence of such counsel we venture the opinion that defective teeth in underweight children of the age range observed are to be regarded as a result of faulty diet and hygiene rather than as a consistent cause for weight deficiency.

Defects of vision did not afford a basis for comparison such as has already been discussed. Occasionally we have been able to trace weight deficiency to a refractive error.

For example, an eight-year-old girl in the underweight group had frequent headaches from eye strain due to hyperopic astigmatism. Because of these headaches she did not take proper amounts of food which, in turn, resulted in weight loss. This girl was fitted with glasses following which her headaches soon disappeared, her appetite improved and weight gain became very satisfactory.

An investigation to determine the extent to which tuberculosis enters as a causal factor in weight deficiency among these children has not yet been completed. So far we have a record of one out of every ten children living in homes where repeated contact has been or is being made with victims of this disease. Six have been previously hospitalized for treatment and at least as many more are definitely pre-tuberculous. We feel quite certain that further search will reveal more tuberculous families and pre-tuberculous children.

So far we have dealt with some of the physical defects which play a part in retarding normal weight progress. Surveys of practice of health habits were, from the beginning, a routine part of the study. In view of the fact that not one of the group was routinely carrying out these habits upon the first survey, our interest became two-fold: (1) in the relation between faulty health habits and weight deficiency and (2) in efforts to bring about their proper practice and noting the effect on weight improvement.

Although the practice of all health habits was encouraged, only those pertaining to food and rest have been considered in this study.

Every child was surveyed once a month and nothing short of a daily routine was considered as "practiced." Neither child nor teacher made these surveys; some member of the staff carried out this detail of work in order that greater uniformity might result.

When observations had been carried on for three months the whole group record revealed considerable variation in the practice of these habits. In order to measure the response in this

time, the first and third records were tabulated and differences computed.

The best response was found to be to the item requiring a mid-morning lunch, including a glass of milk. For this outcome credit is largely due to efforts made by teachers. Of other food habit items, those requiring a cooked cereal for breakfast and drinking four glasses of milk a day showed the greatest increase in observance. This points to home effort, which at the same time was found wanting for items requiring fresh fruit and no tea, coffee, fried or greasy foods.

Of the rest habit items, the greatest increase was noted in that requiring a rest period after school. Evidence of interest and cooperation of parent, teacher and child is shown by the increased observance of rest habits found upon the third survey.

In order to better evaluate the effect upon weight increase of the routine practice of food and rest habits, it was necessary to select as a group those children without physical defects and therefore free to gain.

From the records of this selected group it was found that the best weight gains were made by those observing all habits and the least gains by those deficient in this respect. The average gain for the latter was even less than that for the whole underweight group. Those who practiced habits of rest and not of diet gained more than those who practiced habits of diet and not of rest.

We found that the majority of underweight children with defective tonsils and good records of health habits had poor records of weight progress.

The record of Henry ,K., age 8, and 8 per cent underweight, is cited as a typical example. His health habit record shows good response: an effort to gain, but defective tonsils serve as a handicap. He made no gain in weight in three months and even lost a pound eight months after the original weight record.

It is to be expected that a physical defect and poor practice of health habits would, in most cases, impair weight increase.

Another boy, Horace A., age 11, and 14 per cent underweight has defective tonsils and a poor record of health habits. His daily routine at the time of survey was:

Up at 5:30 A.M.

Helps with chores.

Takes bus to school at 7:30.

Plays at recess time.

No rest period at school.

Home by 3:30.

Plays until supper time, or helps on the farm.

To bed around 8 P.M.

His weight curve showed, after the first two weeks, a decline below the original weight and there remained.

Other children in the rural district with similar weight curves were questioned as to their daily routine and most of them gave a similar account.

The Public Health Nursing Association of Lexington conducts each summer a camp for underweight and pre-tuberculous children. During the past summer 33 of our underweight children attended this camp. It is interesting to note the daily routine which these children were required to follow:

(1) Arise at 6:30 A.M. Bathing and teeth brushing.

(2) Breakfast at 7 A.M., after which, teeth brushing and drinking of water.

(3) Supervised play until 10 A.M., with lunch at that time. Those more than 10 pounds underweight and with rise of temperature rest until noon. Others allowed to play.

(4) At 11:45 wash hands and face for dinner. Dinner at 12. Brush teeth after dinner.

(5) All rest in bed from 1 to 3 P.M., with lunch at that time.

(6) Play until supper time. Temperatures taken at 4 P.M.

(7) At 5:15 wash hands and face for supper. Supper at 5:30.

(8) Story telling from 6 to 8 P.M.

(9) To bed at 8 P.M.

Those children who remained in camp three weeks, or more, made an average gain of 0.68 pound per week as against 0.12 pound average per week for the whole underweight group during the period of this study.

The effect of careful supervision and management on weight gain is further illustrated in the records of a pre-tuberculous boy, Gordon A., who attended this camp for a period of 54 days. His health habit record was at best but fair while in school, but excellent while in camp. His weight curve up to the time he entered camp showed no sustained progress. While he was in camp a decided improvement came about, only to recede after returning home, as found when he entered school in the fall. The camp routine could have been continued at home but it is evident that this was not done.

Although this study is not yet completed, we are lead to draw some inferences with respect to the extent of our future concern in dealing with underweight school children.

It is evident that there are in general two classes of underweight school children. In one belong those to whom we can render little assistance; in the other are those to whom assistance may be given. This service will be of little avail, however, unless it is shared by child, home

and school. The crux of the problem comes in how best to obtain a correlated and effective working of these agencies.

We should bear in mind that not underweight, but its underlying cause, should be searched for and corrected. Little benefit may result from practice of health habits unless the child is rendered free to make a normal gain in weight.

Underweight children afford an opportunity for linking up interest of parents and teachers in the health welfare work of the health department done in behalf of school children. Since we have undertaken this study, less than a year ago, more schools have installed hot noon-day lunches, an increased interest is being manifested in correction of defects, and a check-up on the practice of health habits is to be made a part of the school curriculum.

DISCUSSION (*Abstract*)

Dr. H. S. Mustard, Murfreesboro, Tenn.—Dr. Allen's paper is a report of a series of observations, checked by controls. He has gotten away from the type of report in which conclusions are based upon such weak props as "undoubtedly" and "it seems to me." In our county we have been particularly interested in measures designed to prevent malnutrition, and have studied the relationship of underweight to malnutrition. We have not been satisfied to assume that a child 7 per cent or even 10 per cent underweight is necessarily under-nourished; nor that a child with optimum weight is necessarily well nourished.

In studying the records of examinations of 4693 school children it was found that of those in the "normal weight zone," 83 per cent were rated clinically as being well nourished, 16.3 per cent as having slightly impaired nutrition, and 2.7 per cent as being definitely malnourished. Of that group of children weighing 10 per cent or less than the "normal" for height and age, 17.8 per cent were rated as normal, from a clinical standpoint, 50 per cent as having slightly impaired nutrition, and 32.2 as being definitely malnourished. This finding would tend to substantiate the suspicion that weight and nutrition cannot be used as synonymous terms.

In connection with dental caries, where 2837 children were studied, it was found that in the well nourished and malnourished there was practically no difference in the incidence of this defect. This is too small a group to justify definite conclusions, but the findings suggest that nutritional status bears no immediate relation to dental caries, which is a corollary to the fact that the formation of most of the teeth occurs before school age.

I was interested in Dr. Allen's attitude that the question is not "how much does the child weigh?" but rather "is he maintaining a steady rate of gain?" Such a history or record is important, for our once a year examination can be but a brief cross section of the physical trend of the individual.

Dr. W. D. Beachman, Hattiesburg, Miss.—Has anyone made urinalyses to see what per cent of these children have diabetes?

Dr. Alexander S. Garrett, Weatherford, Texas.—I am of the opinion that the want of milk, failure to drink cows' milk, is an important cause of malnutrition in children and underweights. If the people in general understand the importance of milk for children, and give up tea and coffee, we shall gain a great point in public education.

Dr. Allen (closing).—This is a preliminary study. It was made to separate the wheat from the chaff of interest of the health department in underweight school children. We had to use a small group of children, and therefore could not carry on extensively, and the results are not what we hope for when the work is completed.

We have not yet taken up the question of diabetes in underweights. This and other phases of study we shall engage in this year. These will probably furnish the basis for a report at a future date.

THE DUODENAL TUBE: ITS USE IN SURGICAL UROLOGY*†

By JOSEPH F. MCCARTHY, M.D., *and*
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and
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In 1923 our attention was first attracted to the possibility of utilizing the duodenal tube as a therapeutic measure in post-operative urological cases. The duodenal tube proved to be an efficient means of administering fluids, medications and feedings. It was predicted at that time that the duodenal tube might have a wide field of usefulness in the pre-operative and post-operative treatment of cases showing a relatively acute retention of nitrogenous waste products, and for the prompt relief of severe acidosis. Subsequent experience has confirmed these original predictions.

The object of this preliminary report is not to recount spectacular results accomplished by this method of treatment but rather to illustrate the physiological principles guiding our use of the duodenal tube. In the earlier cases, tubes carrying Einhorn buckets were utilized, but for the more recent cases, Lyon's duodenal tube and the duodenal catheter have been adopted with greater satisfaction. The procedure followed for the introduction of the tube is that described by Lyon.

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*Read in Section on Urology, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

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The duodenal tube not only permits the introduction of feedings and medications, but it can also be used for duodenal drainage. Where it is desired to institute duodenal drainage, the duodenal contents are aspirated when the tip has reached the duodenum. The drainage is initiated by gentle suction applied by a syringe to the end of the tube; a steady flow is obtained of clear golden-yellow fluid, alkaline to litmus but acid to phenolphthalein. When the flow has been started the remainder of the bile is syphoned off. Lyon has found that

"Magnesium sulphate possesses some intrinsic property, hormonic or otherwise, that is not possessed by peptone or the other list of chemicals tried, which favors the evacuation of gall bladder contents, if in no other way than by lowering the duct pressure."

By relaxation of the sphincter of Oddi, following the introduction of the magnesium sulphate (25 c. c. of 33.66 per cent solution) a bile of light golden-yellow color and of medium viscosity like that of syrup is syphoned off. This is known as "A" bile and is obtained from the common duct and may vary in amount from 10 to 30 c. c. The bile suddenly deepens to a darker golden yellow and becomes more viscid, draining either steadily or intermittently until 10 to 300 c. c. have syphoned off. This fraction is called "B" bile and is evacuated from the gall bladder. A second change to a light transparent yellow, thinner and more limpid bile follows. This last fraction Lyon calls "C" bile and ascribes its source to the hepatic ducts. The "C" bile continues to flow with considerably greater intermittency as long as a relaxation of the sphincter is maintained. It is only after these three fractions of the bile have been syphoned off, that biliary drainage can be considered to be complete. From the standpoint of the urologist biliary drainage has a twofold object. (1) Drainage of bile removes from the body nitrogenous waste products. The concentration of the nitrogenous waste products in the bile is dependent upon their concentration in the blood. The value of biliary drainage from this standpoint will be discussed later in presentation of the data of Case[2]. (2) Systematic biliary drainage is the most effective method of draining the liver itself, particularly in patients receiving a very restricted diet by mouth. Moreover any toxic substances drained from the liver are entirely removed from the body, and hence the possibility of their reabsorption into the portal circulation is minimized. Lyon presents abundant evidence to show that biliary drainage enhances liver function. Since the liver is the organ of detoxication par excellence, in the body, it follows that the maintenance of maximum hepatic function is of value in cases presenting symptoms of toxemia.

After the biliary drainage has been completed, the duodenal tip is dropped to the jejunum and feedings, fluids and medications are administered by gravity in solutions warmed to 38° C., at a rate of about sixty drops per minute. For feedings, we find peptonized milk very satisfactory. The nutritive value of milk may be reinforced by the addition of glucose, lactose or olive oil. Enough milk is given to maintain the patient in nitrogen equilibrium. For an afebrile patient at rest, this can be accomplished on a low protein level of 5 to 8 grams per day. Milk is the food of choice, because it is a liquid and its proteins contain the amino acids essential for the body's nutrition. The caloric value of the feeding can be increased by the addition of the glucose, or lactose, or of olive oil. The maintenance of a high carbohydrate intake is desirable for many reasons. Carbohydrate is a body protein sparer. Increasing the carbohydrate intake adds to the liver's store of glycogen and thus improves liver function. Moreover carbohydrate is an anti-ketogenic compound and inhibits the production of an acidosis due to ketogenesis. The addition of lactose to the milk tends to lower the pH of the intestinal contents, retarding protein putrefaction and increasing the rate of absorption of calcium. In the adult there is little if any calcium storage, hence the calcium intake must meet the body's requirements. According to Sherman, the average calcium requirement of the adult is 0.45 gram per day. Since milk contains about 0.20 per cent of the calcium, the total calcium requirement is supplied in 250 c. c. of milk, provided conditions in the small intestines are maintained most favorable to the absorption of the calcium. The tip is drawn back to the duodenum about one hour after the administration of the feeding has been completed and biliary drainage resumed.

Biliary drainage has been adopted as a therapeutic measure only in the more recent cases, originally the tube being utilized for feedings and lavage. Ringer's solution, or physiological salt solution, warmed to 38° C. has been introduced through the tube to increase the fluid intake. When intestinal evacuation is desired, sodium sulphate is added to the solution. The administration of fluids through the duodenal tube has proven a more efficient therapeutic procedure to increase the volume output of urine than intravenous infusions, hypodermoclyses or proctoclyses. Moreover, large volumes of fluid

(12 liters) can be given without discomfort to the patient and in some instances without interrupting the patient's sleep. The evacuation of large volume of fluid by the rectum follows this grand lavage. It has been found that in these liquid feces a greater amount of non-protein nitrogen is eliminated than in the solid feces. This elimination of nitrogen helps to diminish the nitrogen retention in blood, probably because the nitrogenous waste products, for example, urea, excreted by the small intestines are not reabsorbed in the large intestines, as they would be in the absence of the lavage.

Probably the greatest aid to the urologist offered by the duodenal tube is its use in controlling the acid-base balance of the blood. Clinical and biochemical evidence will be presented to demonstrate that alkalies can be administered with greater safety and more efficiently through the duodenal tube than by intravenous infusions, by mouth or by rectum. A striking illustration of this fact was witnessed recently. A post-nephrectomy patient had a carbon dioxid combining power of 21.0 volumes per cent with clinical evidence of a severe acidosis. Fifty cubic centimeters of a 2 per cent solution of sodium bicarbonate were given through the tube every hour for 24 hours. At the end of the 24 hour period, the carbon dioxid combining power was 56.0 volumes per cent. To effect the same result according to Palmer and Van Slyke's formula, 53 grams of sodium bicarbonate would be required by mouth. The administration of the alkali was continued during the night while the patient slept. Our experience has shown that an alkalosis may be readily produced by alkali administration through the tube, consequently the use of the alkali must be controlled at all times by the carbon dioxid combining power of the blood plasma. The administration of acidulated fluids should be checked up for the same reason.

To demonstrate the use of the duodenal tube in surgical urology, clinical and biochemical data are presented on the two following cases:

Case 1.—This was a poorly nourished man of 35 years admitted upon the medical service, complaining of pain in the upper left quadrant of the abdomen and radiating to the left testicle. During previous attacks, the onset of the pain had been accompanied by nausea, vomiting and hematuria. Functional and x-ray studies of the gastro-intestinal tract revealed no pathology. A tentative diagnosis of ureteral calculus was made and the patient was referred to the urological service. Shortly following his admission to our ward March 22, 1923, a pyelographic study was made, the medium employed at that time being a solution of bismuth ammonium citrate. A negative report for calculus, or any observable pathological condition was received but as an apparent direct sequence of this examination pain in the region of the left kidney and along the course of the ureter became very pronounced. The volume of urine excreted diminished and as a signal of impending trouble, severe and repeated nausea and vomiting developed. Practically complete anuria persisted during the fourth, fifth and sixth days after the pyelogram.

To clear up the possibility of a retention of pyelographic fluid in the renal pelvis, an attempt was made to pass a ureteral catheter up the left side but an obstruction was encountered from 6 to 8 cm. from the vesical orifice of the ureter. This obstruction was not met in the previous examination. In the late afternoon of March 30, the advisability of double renal decapsulation was considered, but the majority opinion opposed this procedure on the ground that it was of dubious value. The usual conservative measures of hot packs, hypodermoclyses of 1000 c. c. physiologic sodium chlorid solution were employed. Shortly after this, the patient voided 205 c. c. at one time. The urinary output from March 28 was: 16 c. c.; March 29, 210 c. c.; March 30, 205 c. c.; March 31, 107 c. c.

On March 31, 500 c.c. of blood were withdrawn and 500 c. c. of 3 per cent sodium bicarbonate solution were introduced intravenously. At this time the feasibility of employing the duodenal tube was discussed. This suggestion appealed to one of us as offering the only possible means of assistance since the vomiting had almost reached the projectile stage, and the patient was intolerant to anything by rectum. The twenty-four hours of the day the duodenal tube was first employed, beginning in the late afternoon, the urinary output was 170 c. c. Next day, the output was 330 c. c. Medication employed through the duodenal tube was 5 per cent glucose solution, 2 per cent sodium bicarbonate, and for catharsis sodium sulphate. On the day after, the patient voided 1,055 c. c. and on the next succeeding day 2,250 c. c. For some days prior to this he had the typical appearance of a nephritic, being more or less edematous, with the face rather puffed, palpebral orifices narrowed, edema of the extremities, and twitching and hiccough of a most persistent nature. The feeding through the duodenal tube consisted of peptonized milk, sodium bicarbonate, cream, water and glucose solution, about 1,000 calories every twenty-four hours.

The patient was now in a semi-stuporous condition except when aroused or during the feeding. At other times, he lay with the eyes closed, although his responses were almost always clear. Following this phenomenal improvement in the renal output, April 3 the duodenal tube was discontinued. The patient was put on very small quantities of spinach soup, cauliflower, sweet potato, baked apple and lemonade. The feedings were given in very small amounts. The urinary output during this twenty-four hours was 2250 c. c. This diet was continued another day, and on the evening of April 5, after two days without the tube, the patient had a definite convulsion, lasting fifteen minutes. He was placed in hot packs for twenty minutes, but was seized with another convulsion lasting ten minutes. Chloral and bromids were administered; 500 c. c. of blood was withdrawn and 500 c. c. of 5 per cent glucose solution were administered intravenously about three hours after the last convulsion. Use of the duodenal tube was resumed the following morning, April 6, and continued until April 12. It was introduced at times merely for feeding; at others it was retained for a period of from four to six hours. At one period, it was

left in for forty-eight hours without removal. Later, in the employment of the tube, a feeding would be followed by a six-hour wait, when a half gallon to a gallon of water would be passed through the tube for the purpose of irrigation. Immediately following the onset of the grand lavage, stools increased from two each day to from ten to fifteen. The stools, which at the outset of this procedure gave every evidence of profound putrefactive changes, promptly lost these qualities.

March 27 after the period of oliguria and anuria, we find the urea nitrogen has risen to 62.5 and the creatinin to 12.5 mg. per hundred cubic centimeters. The concentration of sugar in the blood has been doubled, but the chlorids are diminished. Following the suggestion of Myers and Short, the chlorid of the whole blood rather than the plasma have been determined, and these authors place the normal range at from 0.45 to 0.50 per cent. The carbon dioxid combining power shows a beginning decrease in the alkali reserve of the blood plasma. On this date, an obstruction was encountered during an attempt to catheterize through the left ureter. Three days later, March 30, the urea nitrogen has reached 83.3 mg. and the creatinin, 17.0 mg. and twenty-four hours later, these components have increased to 92.0 and 23.1 mg. respectively.

It is evident from a comparison of these figures that the nitrogenous waste products were rapidly accumulating in the blood but all to an equal degree. This fact, as Foster has demonstrated, indicates an anuria of mechanical origin, either in the kidneys or in the ureters. There is, moreover, a precipitous drop in the carbon dioxid combining power to 31.9, March 30 and 21.4 March 31. During this four-day period, about 1.5 to 2 liters of alkaline fluids had been given in influsion, hypodermoclyses and rectal drips. It is significant that, in spite of this large fluid intake, the retention of the nitrogenous waste products progressed and a severe acidosis developed.

There is a well established doctrine stressed by numerous experimentalists and clinicians that cases of nitrogen retention require a large volume of fluid intake to provoke a diuresis since the damaged kidney can excrete the nitrogen compounds at but a low concentration. This principle finds confirmation in the experiments of Foster, who showed that in such cases the amount of nitrogen excreted in the urine bears a definite relation to the volume of urine. It is evident from the blood finding that the introduction of fluid through these channels had little or no influence on the excretion of urine and, consequently, little or none on the retention of nitrogen. The daily volume of urine ranged from 16 to 205 c. c. only one-seventh of the total fluid intake.

On the afternoon of March 31 the duodenal tube was introduced, and through it the administration of fluids, medications and feedings begun. For the subsequent four days, the fluid intake varied from 780 to 3,840 c. c. and the urine excretion rose from 170 to 1,055 c. c. A large quantity of fluid was also lost in the frequent semi-fluid stools passed during this period. It will be noted that although there was a slight rise in urea nitrogen, April 2, to 98 mg. it dropped the following day to 82 mg. and the creatinin was decreased to 20 mg. The influence of this mode of therapy on the alkali reserve of the blood was more striking. Van Slyke, Stillman and Cullen have shown that the normal blood has an alkali reserve equivalent to from 53 to 70 c. c. of carbon dioxid per 100 c. c. of plasma. A decrease of the carbon dioxid combining power below 50 per cent by volume indicates an alkali deficit, or acidosis, whereas an increase above 77 per cent by volume denotes an alkali excess, or alkalosis. The carbon dioxid combining power, April 2, had reached 55.7 and April 3, 81.5.

The drop in the carbon dioxid combining power of the blood plasma from 50.4 to 21.4 within four days demonstrated an extreme depletion of the blood plasma base despite the administration of large amounts of sodium bicarbonate in colonic irrigations, rectal drips and intravenous infusions. Hence, sodium bicarbonate was added to the duodenal lavage, but the use of this alkali was controlled by changes in the carbon dioxid combining power of the plasma. April 2 the alkali reserve of the plasma had risen to 55.7 and April 3 to 81.5 per cent by volume. To avoid the development of an alkalosis, the alkali therapy was discontinued. Since it was thought that the patient had passed the most critical period of his damaged renal function, the duodenal tube was removed, and for the subsequent two days feedings were given by mouth. The night of April 4, the patient had two severe convulsions. A specimen of blood obtained at this time showed that the urea nitrogen had risen to 104 mg. and the creatinin to 21.2 mg. whereas the carbon dioxid combining power had dropped to 63. The duodenal tube was again introduced April 6 and used until April 12, for feedings and lavage. Since the carbon dioxid combining power remained well within normal limits, the use of alkalis in the fluids was abandoned. Tap water alone served for the lavage. April 8 the urine contained several brick-red fragments of a calculus. These were recovered, dried and weighed. The total weight of the fragments was 0.5 grams. They were composed of urate and ammonium phosphate crystals embedded in material of a protein nature.

It is of interest to note that the decrease of the urea nitrogen preceded the drop in the creatinin. A comparison of the relative concentration of the chlorids with that of the nitrogenous waste products and sugar reveals an interesting phenomenon. As the nitrogenous compounds accumulated in the blood, and the sugar rose with them, the concentration of the chlorids progressively diminished. April 4, when the urea nitrogen and sugar had reached the highest levels, 104 mg. and 0.332 per cent, respectively, the chlorids had dropped to their lowest figures, 0.300 per cent. But after this time, the retention of the nitrogenous compounds diminished and the chlorids simultaneously rose to an abnormally high concentration. An explanation of the significance of this balance in the crystalloids of the blood cannot now be presented. However, analogous changes have been observed by us in cases of mercuric chlorid nephrosis.

Case 2.—This is a male, C. M., aged 48 years, admitted to the urological service April 25, of this year. Twenty-five years previous to admission, a left orchidectomy was performed for tuberculosis. On admission the patient complained of dysuria, diurnal and nocturnal frequency, burning during micturition, and of intermittent hematuria for the preceding year. Acid-fast bacilli were found in catheterized bladder specimens for three successive days after admission. Cystoscopy revealed ulceration of bladder, most marked in the region of the

TABLE I.—CHEMICAL CHANGES IN THE BLOOD

DATE	Uric Acid	Urea Nitrogen	Creatinin	Sugar	Chlorids	Carbon Dioxid Combining Power
	mg. per 100 c.c.	mg. per 100 c.c.		Per Cent	Per Cent	
3-13-22	4.6	12.1		0.117	0.487	
3-27-22		62.5	12.5	0.230	0.413	50.4
3-30-22		83.3	17.0	0.246	0.400	31.9
3-31-22		92.0	23.1	0.290	0.357	21.4
4-2-22		98.6	20.4	0.226	0.350	55.7
4-3-22		82.0	20.0	0.270	0.326	81.5
4-4-22		104.0	21.2	0.332	0.300	63.0
4-10-22		56.0	9.2		0.450	56.0
4-15-22		48.0	4.4		0.538	62.6
4-22-22		12.9	3.2	0.141	0.550	60.7
5-4-22		9.7	2.7	0.109	0.513	55.8
7-11-22	4.2	16.0		0.097	0.550	

COMMENT

3-22-22 **Pyelogram.**

3-25-26-22 Oliguria.

3-27-22 Anuria; obstruction encountered about 6-8 cm. from ureteral orifice.

3-28-31-22 Daily urine excretion varied from 16 to 205 c. c. About one-seventh total fluid intake.

3-28-31-22 P.M., about 1,000 c. c. of alkaline fluids were given daily in infusions, hypodermoclyses and rectal drips.

3-31-22 P. M., duodenal tube introduced; solutions of glucose and sodium bicarbonate and tap water given through duodenal tube.

4-1-3-22 Fluid intake, from 780 to 3840 c. c.; daily urine excretion from 170 to 1055 c. c. in 24 hours.

4- 3-22 Alkali therapy discontinued.

4- 3- 4-22 Administrations of fluids and medications and feedings through duodenal tube discontinued.

4- 4-22 P.M. Convulsions.

4- 5-12-22 All fluids and feedings through duodenal tube.

4- 8-22 Calculus passed in urine.

5- 6-22 Patient left Hospital.

right ureteral orifice. Urine obtained by catheterization of the left ureter revealed no organisms. The ureteral catheter encountered an obstruction on the right side. Following an intravenous injection of indigo carmine, no dye was observed from either side during 30 minutes. A phenolsulphonphthalein test, carried out according to procedure recently described by Crowell, (total bladder urine) gave 13 per cent during the first 15 minutes, 12 per cent during the second, 8 per cent in the third, and a trace for the fourth 15 minutes.

Roentgenograms showed a marked tumefaction of the left kidney with fibrotic changes. The right kidney was normal in size and shape but numerous calcareous deposits were noted in the region of the central calyx towards the cortex. Tuberculosis was suggested as the etiological factor.

In a chemical analysis of the blood upon the day following admission, the urea nitrogen was 55 mg. and the creatinin 6.8 mg. per 10 c. c. (*Table II*). A slight acidosis was indicated by the carbon dioxid combining power of 44.7 volumes per cent. During the period of preoperative examinations, due, we believe, to rest in bed and forcing of fluids the urea nitrogen declined to 45.3 mg. on May 6 and to 41.3 mg. per 100 c. c. on May 16. A greater change was noted in the creatinin, which fell to 4.9 and then to 2.2 mg. per 100 c. c. The alkaline reserve of the blood plasma also improved, as indicated by the increase in the carbon dioxid combining power to 55.1 volumes per cent. Throughout this period no variation from the normal was observed either in the sugar or in the chlorids of the blood. In our judgment, the patient at this time was considered to be a fair operative risk and a nephrectomy was indicated. Consequently on May 18 the right kidney was removed. The pathologist reported a caseous tuberculosis of the kidney with hydronephrotic sac formation due to obstruction of the ureter.

For the ten days following operation the volume of urine excreted varied from 1120 to 1830 c. c. However, despite this fair excretion of urine, the patient presented clinical symptoms of renal impairment.

On May 28, the urea nitrogen had risen to 90.6 mg. and the creatinin was 6.8 mg. per 10 c. c. A mild acidosis again had developed with a fall in the carbon dioxid combining power to 43.8 volumes per cent. The duodenal tube was inserted the following day and the regime of lavage and feeding described for the preceding case instituted. Feedings consisting of peptonized milk and glucose, and tap water containing glucose was utilized for the introduction of fluids and for lavage. No alkali was given. During the period of duodenal lavage, the total fluid intake for twenty-four

TABLE II

INFLUENCE OF LAVAGE THROUGH DUODENAL TUBE ON NITROGEN RETENTION AFTER NEPHRECTOMY

Pt. C.M. Age, 48 years. Diagnosis: Renal Tuberculosis.

Date	Urea N.	Creatinin	Sugar	Chlorids	CO_2 C.P.	REMARKS
	Mg. per 100 c. c.				Vol. %	
4-26-27	55	6.8	96	495	44.7	
5- 6-27	45	4.9	132	526	44.7	
5-16-27	41	2.2	.....	.....	55.1	
5-18-27	.....	.....	.....	.....	.....	Right Nephrectomy
5-27-27	90	6.3	87	486	43.8	
5-28-27	.....	.....	.....	.....	.....	Duodenal tube inserted
5-31-27	84	.....	.....	.....	.....	
6- 1-27	80	5.8	89	435	47.5	
6- 6-27	73	4.4	92	396	36.2	
6-10-27	48	7.1	77	462	45.7	
6-13-27	.....	.....	.....	.....	.....	Duodenal tube removed
6-14-27	12	.....	86	562	66.4	

hours averaged from 3,000 to 4,000 c. c. and the urine output varied from 2,100 to 2,800 c. c. This patient was chronically constipated, and required magnesium or sodium sulphate or castor oil through the tube to produce evacuation of intestinal tract. In this respect *Case 2* differs from *Case 1*. In the latter the duodenal lavage was followed by the evacuation of 2 to 10 liquid stools per day. Following the introduction of the duodenal lavage, the urea nitrogen and creatinin of the blood continuously decreased. On June 10 there was noted a sharp rise in the blood creatinin, which was not accompanied by a corresponding rise in the urea nitrogen. Since this followed a rise in body temperature, we believe the increase in creatinin to be due to this fact.

The carbon dioxid combining power on June 6 had declined to 36.2 volumes per cent. To relieve this moderate acidosis, sodium bicarbonate was added to the fluids introduced. A prompt rise in the alkaline reserve of the blood plasma was noted. Subnormal figures for blood chlorids were noted on June 1, (435 mg. per 100 c. c.) and on June 6, (396 mg. per 100 c. c.). However, in this instance the decrease in chlorids is not coincident with a rise in blood sugar as noted in *Case 1*. The blood examination of June 14 gave normal figures for urea nitrogen, sugar and the carbon dioxid combining power but the blood chlorids were above the upper normal level. The hyperchloremia, apparently was due to the secondary anemia. On the following day the tube was finally removed and the patient was discharged improved two days later.

After three months' rest in the country, the patient was readmitted on September 26 complaining of malaise, loss of appetite, oliguria and dysuria. On admission the urea nitrogen of the blood was 136 mg. per 100 c. c. and the carbon dioxid combining power had reached the unusually low level of 18.2 volumes per cent. The relief of this severe acidosis was immediately necessary. The house surgeon immediately dropped the duodenal tube and gave through it four grams of sodium bicarbonate in 2 per cent solution, containing 5 per cent glucose. The following morning the carbon dioxid combining power had risen to 22.1 volumes per cent. At this time an intravenous infusion of 500 c. c. of 5 per cent sodium bicarbonate was administered and this elevated the alkaline reserve to a carbon dioxid combining power of 39.0 volumes per cent. There was noted at this time an increase in the inorganic phosphorus of the blood to 9.9 mg. per 100 c. c. with a decrease in the calcium to 7.7 mg. per 100 c. c. In the meantime a continuous rise in both urea nitrogen and creatinin was noted. A Lyon's duodenal tube was inserted on September 29 and left in place until October 11 except for short intervals when it was withdrawn for cleansing. During this period, no lavage was given. Systematic duodenal drainage, alternating with feedings as previously described, was instituted.

TABLE III.

INFLUENCE OF BILIARY DRAINAGE UPON NITROGEN RETENTION

Pt. C.M. Age 48 years. Diagnosis: Renal Tuberculosis. (After Nephrectomy)

Date	Urea	Creatinin	Sugar	Chlorids	Calcium	Inorg. P.	CO_2 C.P.	REMARKS
	Mg. per 100 c. c. blood				Mg. per 100 c. c. serum		Vol. %	
9-26-27	136		176				18.2	On Admission
9-27-27	172	18.7			7.7	9.9	22.1	
9-28-27	217	20.4	142	519			39.0	
9-29-27	.....	.....	.....	.....	.....	.....	.....	Duodenal tube inserted calcium therapy
9-30-27	132	22.0	114	410	9.8	8.8	54.1	
10-3-27	116	16.8			8.8	6.6	52.2	
10-5-27	95	12.0			7.3	6.3	45.7	Calcium therapy
10-7-27	88	14.0			7.7	5.8	38.1	
10-10-27	88	13.0			7.9	4.5	37.2	
10-11-27					9.6	5.7	35.3	Duodenal tube removed
10-14-27	97	12.8	103	445	9.2	7.3	36.2	

Specimens of bile withdrawn were analyzed for the nitrogenous waste products and data for the results of the biliary analyses are reported in *Table 4,* to be presented later. It is noted in *Table 3* that the continuous biliary (or duodenal) drainage diminishes to some extent the retention of urea nitrogen and of the creatinin of the blood. However, when we compare the influence of biliary drainage upon the retention of the nitrogenous waste products with results obtained by grand lavage in *Case 1* and *Case 2* during the first admission, it is evident that the biliary drainage is of minor importance in the removal of non-protein nitrogenous waste products. Throughout this period of biliary drainage, the patient was chronically constipated, and a fecal evacuation could be obtained only after the administration of castor oil through the tube. Moreover, it is noted that the blood serum calcium tends to fall to a low level, necessitating calcium therapy. Calcium lactate, in doses of 1-5 grams three times a day for 5 days was required to bring the blood serum calcium from 7.3 mg. to 9.6 mg. per 10 c. c. The conclusion seems warranted that conditions in the intestinal tract were not favorable to the absorption of calcium. The initial introduction of alkali through the tube produced a prompt rise in the carbon dioxid combine power of the plasma, but subsequently during the period of biliary drainage the alkaline reserve of the blood plasma continuously decreased to the level of a moderate acidosis.

In *Table 4* are presented the analytical data for the specimens of bile removed in biliary drainage from *Case 2* during a period of 24 hours. A comparison of the concentration of urea nitrogen and of creatinin in the bile drained from the duodenum before the introduction of the magnesium sulphate, demonstrates that the urea nitrogen of the bile is about 69 per cent of that of the blood, but the creatinin is only 20 per cent. Creatinin does not readily diffuse into other body fluids or secretions. This statement is confirmed by the observations of Myers and Fine on the creatinin of spinal fluid, and of Cohen, Killian and Kamner on the creatinin of the ocular fluids. Bile obtained following the introduction of magnesium sulphate contains more urea and more creatinin than the bile syphoned off without this stimulant.

The total amount of bile drained in 24 hours was 1037 c. c. containing 1.229 grams of nitrogen, 1.661 grams of urea and but 0.043 grams of creatinin. The removal of nitrogenous waste product from the body in this way corresponds to the effect of a withdrawal of a similar volume of blood in a phlebotomy, but it is only about one-tenth the amount of nitrogen that would be eliminated in a corresponding volume of urine. Since grand duodenal lavage markedly increases the volume of urine excreted, it is evident that duodenal lavage with large volumes of fluid should relieve nitrogen retention more effectively than biliary drainage. That is in reality what we have found to be the case.

TABLE IV.

REMOVAL OF NITROGENOUS WASTE PRODUCTS IN DUODENAL DRAINAGE.

Pt. C.M. Age 48 yrs. Diagnosis: Renal Tuberculosis (after Nephrectomy)

Time	Volume c. c.	N-P-N	Urea N	Creatinin	N-P-N	Urea	Creatinin	REMARKS
	Mg. per 100 c. c.				Mg. Removed per Volume			
			116.6	17.6				Blood
9:00 to 10:05 A.M.	130	105.6	75.7	3.3	137.3	210.5	4.2	Before $MgSO_4$
11:15 A.M.								25 c. c. 50% $MgSO_4$
11:15 to 1:00 P.M.	110	129.2	107.1	4.0	142.1	252.1	4.4	
3:15 to 5:00 P.M.	145	117.1	98.6	4.6	169.8	306.0	6.6	
6:30 to 7:00 P.M.	200	128.1	62.4	3.7	256.2	267.0	7.4	
8:00 to 9:00 P.M.	102	135.1	79.7	2.9	137.8	174.0	3.0	
	350	110.3	60.3	5.0	386.0	451.5	17.5	During night
Total 1037 c. c.					(1,229 gm. N-P-N. (1,661 gm. Urea (0,043 gm. Creatinin			

SUMMARY

The duodenal tube has in general been confined in the past to a limited field of usefulness in obtaining material for analytical purposes by gastro-enterologists and in the treatment of gall bladder and gastro-intestinal disease. To the urologist it affords a practical means of accomplishing speedily desired results, particularly in the treatment of cases with renal impairment. Biliary drainage through the tube is a time-consuming process, producing little demonstrable effect upon nitrogen retention. However, the patients appear clinically benefited by systematic drainage. This improvement can be attributed to an improvement in liver function. When relief of the nitrogen is urgent, lavage through the tube is far more efficient. Lavage with large volumes of fluids increases the volume of urine excreted and promotes the frequent elimination of liquid feces. Nitrogen is carried off by both channels. Feedings can be conveniently given through the tube maintaining the patient's nutrition at a high level; these feedings and lavage may be continued while the patient is sleeping. The feedings should be regulated to keep the patient at least in nitrogen equilibrium and furnish the requisite amount of calories. Moreover, the tube is a practical means of introduction of medications, particularly for the relief of nephritic acidosis. By means of the tube, the acid-base balance of the blood may be regulated with almost mathematical precision. We are inclined to regard the intestinal tract, as a tube solely designed for digestion and absorption. In addition to this function, the intestinal tract has excretory functions, and these excretory functions can be increased to the maximum by lavage, because the rapid passage of fluids through the intestines prevents the reabsorption of the excretory products. Furthermore, lavage diminishes intestinal putrefaction and the bacterial flora of the intestinal tract can be changed almost at will by the use of the duodenal tube. In this respect, duodenal lavage is more efficient than colonic irrigations. This fact was demonstrated forcibly in *Case 1.* Before the introduction of the lavage the patient was passing a small amount of solid, dark-colored, foul smelling feces, of an alkaline reaction. After 24 hours of lavage, the feces had become light brown in color, liquid and possessed an acid reaction and no perceptible odor. A similar change in the character of the feces could be produced in no other way.

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DISCUSSION (*Abstract*)

Dr. Clarence G. Bandler, New York, N. Y.—For some time I have been convinced of the great utility of the duodenal tube in some urologic surgical conditions, and have had numerous opportunities to observe its practical application. Very frequently as Dr. McCarthy showed in these cases, with pronounced nitrogen retention the most urgent need is the immediate relief of the acidosis. Before we can intelligently employ therapeutic agents to readjust the acid-base balance to normal, it is essential to understand the mechanism of the production of the acidosis. In patients, such as those mentioned by Dr. McCarthy, two factors operate to produce the acidosis, (1) retention of phosphates and (2) ketogenesis.

Marriott and Howland first demonstrated that the acidosis of nephritis was due to a failure of the kidneys to excrete phosphates. This retention of phosphate produces an increase in the inorganic phosphorus of the blood serum. When the phosphates are retained in the blood, the acid phosphates are neutralized by the blood plasma bicarbonate, and are converted into alkaline phosphates. This increase in alkaline phosphates diminishes the calcium ion concentration of the blood serum, since calcium phosphate is formed and may be eliminated by the intestines. There is then noted a decrease in the blood serum calcium, and when this drops to 7.0 mg. per 100 c. c. or less, spasms and convulsions are produced. In a recent communication in collaboration with Dr. John A. Killian, which was presented before the American Urological Association, we emphasized the necessity of following the carbon dioxid combining power of the blood plasma, and the inorganic phosphorus and calcium of the blood serum, and we stressed the point that in the treatment of this type of acidosis our therapy must be directed towards maintaining the blood calcium within normal limits, particularly the ionized fraction of the blood serum calcium. Giving alkali alone will tend to reduce the calcium ion concentration. The administration of calcium is necessary to make up for the decrease in blood serum calcium. Grollman has shown that calcium chlorid added directly to blood diminishes the filtrability of the phosphorus, and consequently decreases the excretion of phosphorus in the urine. Marshall has recently discussed this question in his review, "The Secretion of Urine." Hence, the intravenous administration of calcium is inadvisable. The duodenal tube offers a most efficient means for calcium administration. We find that a convenient calcium vehicle is milk. Cow's milk contains about 0.13 per cent of calcium. Calcium is absorbed from the intestines in a slightly acid medium, in which the calcium compound is ionized. The addition of lactose to the milk promotes the growth of lactic acid producing bacteria, increasing the hydrogenion concentration of the intestinal contents. The existence of such conditions in the intestinal tract promotes the absorption of the calcium. The caloric value of the feedings can be

enhanced by the administration of cod liver oil along with the milk. The calcium of the milk may be increased by adding lime water to the milk, but the reaction of the milk must be kept slightly acid to avoid precipitation of the calcium. By this method of calcium administration we avoid the dangers of hypercalcemia, because it is not possible to increase the blood serum calcium above the normal when the calcium is absorbed from the intestinal tract.

Ketogenesis results when the ketogenic substances, that is, the fatty acids, are oxidized in the body in excess of the carbohydrates oxidized. Ketogenesis may produce an acidosis in patients with impairment of renal function, since the damaged kidneys are not able to produce enough ammonia for neutralization of the keto-acids. This type of acidosis is frequently observed in post-operative cases and in emaciated patients, or after persistent vomiting, because it adds another type of acidosis to the existing nephritic acidosis. Woodyatt has shown that when fatty acids and glucose are oxidized in equimolecular amounts, 1.5 fatty acid to 1 of glucose, no ketogenesis results. The proper balance between ketogenic and anti-ketogenic substances to avoid ketogenesis is expressed in this formula: Fat oxidized may be = 2 carbohydrate + 0.5 protein. The average composition of cow's milk is, fat = 3.0 per cent, lactose = 4.5 per cent, protein = 1.5 per cent. According to this formula we can give about 7.0 gm. more of fat for every 100 c. c. of milk. This is an important factor, since 1 gm. of fat yields 9.5 calories, whereas 1 gm. of carbohydrate or protein yields only 4.1 calories. The most convenient form of fat to be added is olive oil. When additional fat is added to the milk care must be taken to regulate the ketogenic-anti-ketogenic balance.

The duodenal tube has proven to be the most practical method of increasing the fluid intake, of promoting intestinal lavage, of regulating the reaction of the intestinal contents, of regulating the acid-base balance of the blood with any desired precision, and of maintaining an adequate food intake, according to the caloric requirements of the individual, and balanced in respect to its ketogenic and anti-ketogenic factors.

Dr. William A. Frontz, Baltimore, Md.—The duodenal tube, as pointed out, would seem to have three fields of application: first, as a method for giving large amounts of fluid; second, as a means of giving food and, third, as an avenue through which cathartics can be administered. It has been repeatedly demonstrated that many of these patients do splendidly after being given large amounts of fluid. These are kidneys in which there is always a bilateral lesion, and in which there is retained a considerable increase of nitrogenous waste products in the blood stream. The kidney is unable to concentrate, and the substances which are readily handled by the healthy kidney can be eliminated in disease only in considerable dilution.

As Dr. McCarthy pointed out, the ways in which these fluids are usually given, by hypodermoclysis and the intravenous route, may be exhausted, and I think the duodenal tube offers an excellent means of giving fluid easily and in large amounts.

To me, a more appealing thing about the method is the ability to give food in cases of acidosis. It obviates an annoying feature observed not uncommonly after the intravenous administration of glucose; namely, the marked febrile reaction which in our experience has followed the intravenous use of glucose in many cases. Cathartics can also be administered and the intestinal tract emptied, protecting the kidney thereby. This is of marked advantage for the kidney is in very poor shape for handling any increased work. I think the method is a distinct contribution, and I shall certainly use it.

Dr. Albert Goldstein, Baltimore, Md.—We have all been confronted with such conditions as Dr. McCarthy reports, which in most instances have resulted in death because of our inability to combat the existing toxemia. We have all had patients who, following an operation or prior to one, have had distressing symptoms, due to gaseous distension or regurgitated intestinal fluids, or who suffered with pronounced nausea, hiccoughing, and so forth. We all know how important it is that fluids should be forced in the practice of genito-urinary surgery. This, we feel, has a two-fold purpose: first, to relieve the patient of the distressing thirst which comes on either before or after an operation and, second, to produce an increased urinary output, so that the retention products, urea and so on, can be eliminated in greater quantities, thereby improving the kidney function. The use of the duodenal tube in our hands has been very limited, but attended with excellent results.

The duodenal tube has relieved for us a condition that we were unable to check otherwise, namely: persistent vomiting and hiccoughing, and we feel that without it the patients would have died. The duodenal tube can do the following things for us: (1) relieve conditions due to gas and regurgitated intestinal fluids; (2) relieve the nausea and hiccoughing that occur either before or after an operation; (3) make possible the free introduction of water, and thereby relieve the distressing symptom of thirst; (4) permit transgastric feeding; (5) relieve toxemia; and (6) supply a port of entry for all kinds of medication.

In the cases in which we have employed the duodenal tube it was used as a last resort, to check hiccoughing and vomiting, and we did not expect very good results. The only explanation we could offer at the time was that the condition was due to the absorption of the urinary products. No attempt had been made to study the condition more thoroughly, for we were ignorant as to how to go about it. Similar cases had been treated before without the tube, but as a rule they died, particularly when the hiccoughing appeared that sometimes follows a suprapubic prostatectomy.

Dr. McCarthy, in his study, has found a definite explanation of the whys and wherefores in these cases, and I feel as he does, that there is a definite place for the duodenal tube in genito-urinary surgery. It is interesting to note his association with a biochemist in this particular study, a phase probably not appreciated by all of us. Especially am I interested in the early introduction of the duodenal tube, before conditions become too extreme, and, furthermore, in its prophylactic use, in which capacity I am employing it. We certainly are indebted to Dr. McCarthy for bringing this procedure before us, and I feel sure it will have a wide application in the future.

Dr. C. E. Burford, St. Louis, Mo.—Einhorn introduced his first duodenal tube in 1911, and was the first to use it for feeding purposes. When Dr. McCarthy announced to the profession the cysto-urethroscope which bears his name I thought it just another thing from the "effete East" until my friends convinced me of its value, since when I have become a McCarthy convert and started early on the use of the indwelling duodenal tube.

In one case of paralysis of the throat in which the

duodenal tube has been used continuously for six months the nutrition has not only been maintained but the body weight has been increased. We have found that the patients are more comfortable with the tube introduced through the nares than through the mouth. The different uses for the tube have been mentioned; relief from vomiting; relief from toxemia from the small intestine, and its use for feeding. In our experience its use for feeding has been the most important, and the washing out of toxins from the small intestine ranks next in value.

During the past two weeks we have had a frail patient, with both ureters blocked with stones, and persistent vomiting of all water and food for ten days. We removed the left ureteral stone by extraperitoneal open operation but the vomiting persisted and loss of weight continued. The use of the retained duodenal tube made the patient comfortable at once and liquid nourishment is given and retained at regular intervals. The great advantage of the duodenal tube lies in the fact that it can be used simultaneously with transfusion, intravenous glucose, hypodermoclysis and proctoclysis adding a fifth aid to the four great supports of the worried urologist.

A few years ago the general surgeons pointed out the fact that patients with peritonitis do not die of that disease but do die of toxemia of the small intestine, and they were recommending jejunostomies for its relief. Dr. McCarthy's contribution is not only to urology but he has made a contribution which will in some measure repay the general surgeons for what they have taught us in the past.

Dr. Gordon Craig, Sydney, Australia.—Experimental work by more than one man has shown that the intestinal contents of the duodenum of a dog in which an artificial obstruction had been produced, when injected intravenously in relatively small amounts, can produce death in a normal dog. In uremia, there is frequently distention of the bowel amounting to obstruction. Dr. McCarthy has shown how this condition can be relieved by drainage with a duodenal tube.

Dr. Edward L. Keyes, New York, N. Y.—The duodenal tube is a life-saver under the circumstances in which Dr. McCarthy employs it. I have used it and believe I have saved lives thereby which otherwise could not have been saved.

Dr. McCarthy (closing)—The tube is of great use in nephrectomies. I think urology has developed more and more into a biochemical science. I believe we should all familiarize ourselves with biochemical terminology. The use of the tube does not preclude the use of other methods, such as hypodermoclysis. As Dr. Keyes has said, its use is justified for the reason that it will save a life occasionally that could not have been saved before. It is well to know that in a tube-drained patient the treatment may very well be continued at home.

The point I did not call to your attention Dr. Craig emphasized; that is, the rate of absorption in the small intestine. That is a deleterious factor and also an advantageous factor, in that absorption takes place more rapidly in the small intestine and that prompt elimination and medication may be effected by this tubal method.

CHOLECYSTITIS AND CHOLELITHIASIS IN CHILDREN*

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Gall bladder disease in children is commonly thought to be of exceedingly rare occurrence, amounting to a clinical or pathological curiosity, and therefore of slight practical importance. Most text books on pediatrics and surgery make little or no mention of the condition. However, the increasing number of reported cases and comment in the current literature of the past few years indicates that gall bladder disease in children, while not a common condition, deserves consideration. The radiographic demonstration of gall stones in a child, age three years, which the writer was so fortunate as to accomplish, suggests that the condition may merit the attention of the radiologist.

In May, 1925, Dr. J. H. Hartman, of Jacksonville, Fla., referred to me a male child, Horace Lee Golden, age three years and eight months, to confirm an x-ray examination made elsewhere, said to show a right renal stone. My examination revealed a number of faceted stones in the gall bladder region which I reported as gall stones. A second small stone was noted in the liver area, thought to be an intrahepatic or hepatic duct calculus.

These findings were confirmed at the time by Drs. Cunningham and Shaw, of Jacksonville, and again confirmed by them sixteen months later, preceding operation by Dr. Hartman. The gall bladder was removed. The walls were thickened and it contained five small stones, but no bile. There was an apparent atresia of the cystic duct.

The clinical record of the child is of interest, as it suggests that grave consequences resulted from the failure to consider and properly treat the condition at an even earlier age.

The patient is an only child of healthy parents; delivery was normal after four hours' labor. It is not known whether pituitrin was used. Two days after birth the infant had convulsions, frequently repeated for two days. For the following two years he was well and showed normal development, learning to walk and talk. At about the age of two and one-half years he was sick for one week, having fever, nausea, vomiting, abdominal pain, convulsions, and eventually he became unconscious. He recovered, but thereafter had marked left spastic hemiplegia, right facial paralysis and aphasia. These conditions persist to the present date, but have shown improvement with age. Following this severe illness, convulsive seizures have been frequent. The parents describe "stomach attacks," however, which are distinct from the convulsions, although often preceding them. These have been characterized by nausea, occasional vomiting, right upper abdominal pain and the

*Read in Section on Radiology, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

TABLE OF CASES ON CHOLECYSTITIS and CHOLELITHIASIS in CHILDREN up to FIFTEEN YEARS OF AGE

No. of Case	Date	Author	Sex	Age	Autopsy	Operation	Stones	Jaundice	Cholecystitis	Other Pathology	Other Symptoms	How Diagnosed
1	1835	Husson[1]	———	8 yrs.	Yes	No	No	———	Yes	Perforation of gall bladder; with peritonitis	Typhoid fever; death in third week	Autopsy
2	1852	Archambault[1]	———	Infant	Yes	No	———	———	Yes	Perforation of gall bladder; obliteration of common duct	Typhoid fever	Autopsy
3	1853	Barthey and Pilliet[1]	———	12 yrs.	Yes	No	———	———	Yes	Rupture of gall bladder with abscess formation	Typhoid fever; death in fifth week	Autopsy
4	1864	Chedevergne[1]	———	15 yrs.	Yes	No	———	———	Yes		Typhoid fever; death on fifteenth day	Autopsy
5	1882	Stedman[1]	Fml.	13 yrs.	Yes	No	No	———	Yes	Perforation of fundus of gall bladder; peritonitis typhoid lesions	Typhoid fever, complicated by peritonitis	Autopsy
6	1882	Stedman[1]	Fml.	6 yrs.	Yes	No	No	———	Yes	Three perforations in fundus of gall bladder; peritonitis	Typhoid fever, with symptoms of peritonitis	Autopsy
7	1886	Bond[1]	———	10 yrs.	Yes	No	———	———	Yes	Empyema of gall bladder	Typhoid fever with symptoms of pneumonia and meningitis	Autopsy
8	1893	Chiari[1]	———	12 yrs.	Yes	No	———	———	Yes	Rupture of gall bladder; peritonitis due to typhoid bacillus; broncho-pneumonia	Typhoid fever complicated by pneumonia	Autopsy
9	1896	Alexieef[1]	———	5 yrs.	———	Yes	———	———	Yes	Perforation of gall bladder; peritonitis	Typhoid fever; symptoms of acute cholecystitis; distention of gall bladder; acute abdominal pain and distention with disappearance of enlarged gall bladder	Symptoms and operation
10	1898	Wendel	Fml.	19 mos.	Yes	No	Yes	Sclera Yellow	Yes	Perforation of gall bladder; abdominal adhesions; Sero-purulent fluid in abdomen	Diarrhea; vomiting; pain; abdominal distention; fullness in right hypochondrium	Autopsy
11	1898	Wendel	Mle.	7 yrs. 4 mos.	———	Yes	Yes	No	Yes	Perforation of gall bladder; sero-purulent fluid in abdomen	Attacks of abdominal pain and vomiting; abdomen distended and tender; diffused hardness from anterior border of liver to median line; fever	Symptoms and operation
12	1898	Wendel	Fml.	6 yrs.	———	No	Yes	Yes	———		Abdominal pain and distention; vomiting; fever; palpable gall bladder	Symptoms and passage of stones in feces, followed by relief of symptoms
13	1908	Ashurst[1]	Mle.	12 yrs.	Yes	Yes	———	———	Yes	Perforation of gall bladder; peritonitis	Typhoid fever; abdominal pain and signs of peritonitis	Autopsy
14	1910	Prince[1]	Fml.	14 yrs.	———	Yes	No	No	Yes	Clotted bile in gall bladder; motile bacillus isolated	Typhoid fever; pain in gall bladder region; tenderness; marked spasm of upper right rectus muscle	Symptoms and operation
15	1910	Cole and Winthrop	Fml.	15 yrs.	———	Yes	Yes	No	Yes		Typhoid fever 5 yrs. previously; gall stone colic; fever; distention, muscle spasm and tenderness over costal margin in front; tenderness in upper right quadrant	Symptoms and operation
16	1910	Gleeson	Fml.	1 yr. 9 mos.	———	No	Yes	Yes	———	Catarrhal jaundice after gall stone passed	Severe abdominal pain; vomiting; prostration; cold sweats; fever	Symptoms; passage of stone in feces with relief of symptoms

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No. of Case	Date	Author	Sex	Age	Autopsy	Operation	Stones	Jaundice	Cholecystitis	Other Pathology	Other Symptoms	How Diagnosed
17	1914	Downes	Fml.	13 yrs.	.....	Yes	Yes	.....	.....		Acute symptoms suggesting appendicitis	Operation
18	1914	Pybus	Mle.	6 yrs.	.....	Yes	No	No	Yes		Abdominal pain, right side; distention right side; rigidity and tenderness, upper part of right rectus and slight over right iliac fossa; fever	Operation
19	1918	Guttfeld	Fml.	15 yrs.	.....	Yes	Yes	No	No		Abdominal pain; general tenderness; rigidity on right side; bile pigment in urine	Operation
20	1919	Deaver[1]	Fml.	5 yrs.	.....	Yes	No	.....	Yes	Necrotic area in fundus of gall bladder	Typhoid fever; severe abdominal pain with signs of peritonitis	Operation
21	1919	Marton	.....	2½ yrs.	.....	Yes	No	No	Yes	Stenosis of cystic duct	Symptoms of intestinal obstruction: sausage like mass in right hypochondrium	Operation
22	1920	Linder	Fml.	15 yrs.	.....	Yes	Yes	No	Yes	Diseased appendix at previous operation	Abdominal cramps; palpable tumor in right hypochondrium, very tender	Symptoms and operation
23	1920	Bell	Fml.	14 yrs.	.....	Yes	Yes	No	Yes	Appendix contained enterolith	Abdominal pain locating in right upper quadrant; tenderness and rigidity on right side; vomiting; fever; leucocytosis	Symptoms and operation
24	1920	Reed and Montgomery	Mle.	11 yrs.	.....	Yes	No	No	Yes	Beginning necrosis in gall bladder wall; clumps of bacilli in contents	Typhoid fever; severe abdominal pain; visible distention of gall bladder; tenderness and muscle spasm in this region	Symptoms and operation
25	1920	Reed and Montgomery	Fml.	12 yrs.	.....	Yes	Yes	No	Yes		Typhoid fever 8 mos. previously; acute abdominal pain; signs of gall bladder infection; indefinite mass; fever; leucocytosis	Symptoms and operation
26	1920	Reed and Montgomery	Fml.	13 yrs.	.....	Yes	No	No	Yes	Pus from gall gladder contained typhoid bacilli	Typhoid fever; abdominal pain; tenderness in epigastric and right hypochondriac region; rigidity right rectus muscle	Symptoms and operation
27	1920	Reed and Montgomery	Mle.	9 yrs.	.....	Yes	No	Yes	Yes		Typhoid fever; acute abdominal pain; distention in right upper quadrant; palpable gall bladder; muscle rigidity	Symptoms and operation
28	1920	Reed and Montgomery	Mle.	15 yrs.	.....	Yes	No	No	Yes	Slight inflammation of appendix; distention of gall bladder	Typhoid fever with symptoms of intestinal perforation	Operation
29	1920	Reed and Montgomery	Fml.	8 yrs.	.....	Yes	No	No	Yes	Marked distention of gall bladder; typhoid bacilli in cultures; necrotic areas in gall bladder walls	Typhoid fever just previously; abdominal pains; asymmetrical abdominal distention with bulging on right side; visible and palpable mass on right side; fever	Operation
30	1920	Friedenwald and Ullman	Fml.	15 yrs.	No	Yes	Yes	No	.....		Right abdominal pain; tenderness right hypochondrium; slight fever and chills; vomiting	Symptoms and operation
31	1921	Freundlich	.....	2 mos.	Yes	No	Yes	.....	.....	Premature infant	Fever; abdominal distention	Autopsy

TABLE OF CASES ON CHOLECYSTITIS and CHOLELITHIASIS in CHILDREN up to FIFTEEN YEARS OF AGE

No. of Case	Date	Author	Sex	Age	Autopsy	Operation	Stones	Jaundice	Cholecystitis	Other Pathology	Other Symptoms	How Diagnosed
32	1922	Houze[3]	———	5 yrs.	.....	.....	No	.....	Yes			
33	1924	Fischer	Fml.	9 yrs.	.....	Yes	Yes	No	Yes		Abdominal pain; fever; vomiting; tenderness and rigidity at costal margin on right side	Operation
34	1924	Fischer	Fml.	11 yrs.	.....	Yes	No	No	Yes	Typhoid bacilli in gall bladder contents	Typhoid fever; severe pain; tenderness, rigidity and some distention in right upper quadrant	Symptoms and operation
35	1924	Fischer	Fml.	2½ yrs.	Yes	Yes	No	No	Yes	Enlargement of lymph glands in the region of the head of the pancreas compressing choledochus	Scarlet fever; abdominal pain, distention, tenderness and rigidity in right upper quadrant; palpable gall bladder	Operation and autopsy
36	1924	Fischer	Fml.	11 yrs.	.....	Yes	No	No	Yes	Staphylococcus pyogenes in gall bladder contents	Fever; chills; abdominal colic; vomiting; some abdominal distention; marked rigidity and tenderness in right upper quadrant	Symptoms and operation
37	1925	Paterson and Wyllie	Fml.	9 yrs. 5 mos.	.....	Yes	Yes	No	Yes	Slightly inflammed appendix removed at previous operation	Abdominal pain; vomiting; some tenderness over right half of abdomen	Operation
38	1925	Key	Fml.	15 yrs.	.....	Yes	Yes	No	Yes		Abdominal pain; chiefly epigastric, symptoms suggesting appendix in high position	Operation
39	1925	Joll	Fml.	14½yrs.	.....	Yes	Yes	Yes	.....		Abdominal pain, biliary colic type; tenderness on pressure over right hypochondrium	Symptoms and operation
40	1925	Palsey	Mle.	12 yrs.	.....	Yes	Yes	Yes	Yes	Ascaris in centre of one gall stone	Abdominal pain; fever; palpable swelling	Symptoms and operation
41	1925	Hamerton	Fml.	3 yrs.	.....	Yes	No	No	Yes		Abdominal pain and vomiting; abdomen distended; rigidity and tenderness in right upper quadrant	Operation
42	1925	Kennedy	Fml.	13 yrs.	.....	Yes	Yes	No	.....	Concretion and worms in appendix	Abdominal pain and vomiting; right lower rectus somewhat rigid	Operation
43	1925	Edington	Fml.	15 yrs.	.....	Yes	Yes	No	Yes		Pain in abdomen and back; vomiting; rigidity of upper right rectus; tenderness near McBurney's point	Operation
44	1925	Gehwolf	Mle.	8 yrs.	No	Yes	Yes	Yes	Yes	Stenosis of cystic duct	Abdominal pain and vomiting; rigidity; tenderness at McBurney's point and in right upper quadrant	Operation
45	1925	Snyder	Mle.	4 yrs.	No	Yes	Yes	.....	Yes	Appendiceal adhesions	Pain, rigidity, vomiting, fever leucocytosis	Operation
46	1925	Snyder	Mle.	5½ yrs.	No	Yes	No	No	Yes	Acute appendicitis	Pain, vomiting, rigidity, fever leucocytosis	Operation
47	1925	Snyder	Mle.	9½ yrs.	No	Yes	No	No	Yes	Appendix moderately inflammed	Pain, vomiting, rigidity, fever, leucocytosis	Operation
48	1925	Kellogg	Fml.	15 yrs.	No	Yes	Yes	No	Yes	Peritonitis due to perforation of gall bladder	Sudden, severe general abdominal pain; board like rigidity; vomiting; fever; leucocytosis	Operation
49	1925	Peterson	Mle.	8 yrs.	.....	.....	.....	.....	Yes			
50	1925	Peterson	Fml.	15 yrs.	.....	.....	Yes	———	Yes			

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No. of Case	Date	Author	Sex	Age	Autopsy	Operation	Stones	Jaundice	Cholecystitis	Other Pathology	Other Symptoms	How Diagnosed
51	1925	Lilienthal	Fml.	11 yrs.	.....	Yes	Yes	.....	Yes			Operation
52	1925	Costa, S., and Boyer, L.	Fml.	Over 6	.....	.....	.....	.....	Yes	Ascites	Typhoid fever	
53	1925	Judd, E. S.	Mle.	8 yrs.	No	Yes	Yes	No	Yes	Stricture or stenosis of duct	Attacks of severe upper abdominal pain since age 3; x-ray urinary tract negative	Operation
54	1926	Muller	.....	4 yrs.	.....	Yes	No	No	Yes	Serous exudate in peritoneum	Abdominal pain chiefly on right side; vomiting; some abdominal distention; tenderness most marked on right side; under anesthesia, a palpable tumor in right upper quadrant	Operation
55	1926	Brown	Fml.	9 yrs.	.....	Yes	Yes	No	Yes	Congested appendix	Abdominal pain; vomiting; tenderness more pronounced on right side	Symptoms and operation
56	1926	Clifton and Landry	Fml.	11 yrs.	.....	Yes	Yes	No	Yes	Acute appendicitis	Abdominal pain; vomiting; fever; tenderness, slight distention, rigidity in region of gall bladder and over McBurney's point	Symptoms and operation
57	1926	Zeligs	Fml.	12 yrs.	.....	No	No	No	Yes	Typhoid bacilli in bile	Pain, tenderness and rigidity over gall bladder, relieved by injection of mercurochrome and non-surgical biliary drainage	Symptoms
58	1926	Carey	Fml.	9 yrs.	No	Yes	Yes	No	.....	Chronic appendicitis slight inflammation	Right abdominal pain; vomiting; slight rigidity	Operation
59	1926	Yoshio Kishi, J.	.....	46 dys.	Yes	.....	Yes	Yes	.....	Syphilitic	Many convulsions since birth	Autopsy
60	1927	Beals	Mle.	3 yrs. 6 mos.	No	Yes	Yes	No	Yes	Stricture common duct	Spastic paralysis and aphasia	Operation

NOTES:—

1—Cited by Reed and Montgomery. Original articles not available.

3—Houlze's article not available; data from title.

absence of fever. They have been less frequent and severe since the operation, now fourteen months ago. At no time has jaundice been observed.

Manifestly, the convulsions soon after birth may have been due to passage of congenital stones. The grave illness at the age of two and one-half years, which has left him a cripple and incapable of speech, is suggestive of an attack of acute gall bladder disease. Undoubtedly he has been relieved, though not cured, of painful "stomach attacks" by cholecystectomy.

In 1923, Kellogg[35] was able to collect 64 cases reported in the literature of gall bladder disease occurring in children up through the age of fifteen years. These included one of his own cases. These he tabulated, showing the author, sex, age and clinical, operative and autopsy findings. I have been able to collect sixty additional cases. These include the cases reported by Wendell[27] in 1898 and the typhoid cases listed by Reed and Montgomery[25] in 1920 which were omitted by Kellogg; also the one case reported at this time, which appears to be the only case having a preoperative radiographic diagnosis. For purposes of comparison I have tabulated them in keeping with the form used by Kellogg.

A study of reported cases indicates that they may be placed into three groups, as suggested by Kellogg: gall stones in early infancy; gall stones in later childhood; and acute cholecystitis.

Gall stones in early infancy are apparently quite rare, if autopsy records are dependable. However, Wendel says that during one year he found gall stones once in six autopsies upon infants and quotes Valleix to the effect that they are quite frequently found in the newborn. Probably the gall bladder has not been carefully searched for small concretions in many of the routine autopsies reported. Those found at birth or within the first few days are undoubtedly of congenital origin, as may be true also of those reported in the early months. They, very probably, are not of infectious origin. It is believed by Still[36] that their formation is favored by stagnation of bile in fetal life and early infancy. In view of our increasing knowledge of the importance of cholesterol metabolism in the etiology of gall stones, it seems to me conceivable that the hypercholesterolemia of pregnancy may be a predisposing factor in the formation of fetal stones.

The significance of their occurrence lies in the troublesome consequences of later months or years. Occasionally they may obstruct the common duct, which accounts for some of the cases of fatal icterus of the newborn.

Gall stones and cholecystitis occurring in children past early infancy constitute a group which is difficult of diagnosis, due largely to the failure to suspect these conditions in children.

Wendel's article is worthy of study as to the symptomatology. He believes that very young infants have gall stone colic, expressed symptomatically by paroxysms of crying, not quieted by ordinary measures. The attacks may last several hours, alternating with periods of repose. The urine may be of darker color. The feces should be very carefully searched for the small stones.

Older children appear, from the reported cases, to have frank gall bladder attacks with symptoms as characteristic as do adults. The masked gall bladder disease of adults, with symptoms chiefly of dyspepsia, seems to occur in a smaller proportion, or is more often overlooked.

Acute cholecystitis in infants and children may be divided into two groups; the first constitutes a complication of typhoid fever or rarely scarlet fever. These are dealt with in the articles by Reed and Montgomery,[25] Deaver[37] and Fischer.[8] The second group is usually the acute outcome of previously existing chronic cholecystitis, often with stone. As was pointed out by Snyder,[26] the diagnosis may be mistakenly that of acute appendicitis, or appendicitis may be associated. However, the symptoms are sufficiently imperative to lead to operation. The surgeon will not then fail to recognize the condition if the knowledge that acute cholecystitis and cholelithiasis do occur in children prompts him to extend his exploration to the gall bladder.

The frequency of occurrence of gall bladder disease in children, though not great, is probably more than indicated by the reported cases. Alvarez, Meyer, Rusk, Taylor and Eastand[38] believe that many cases diagnosed in adults have their inception in childhood. Mentzer[39] found "cholesterosis" or strawberry gall bladder in one case, age thirteen, out of sixty-three consecutive autopsies (1.5 per cent) on children up to the age of fifteen, and expresses surprise at the high percentage found in young persons. Wendel's article is based on observations of sixteen cases. Farr[40] believes cholecystitis in children is not very rare.

Indeed, it is surprising that cholecystitis and cholelithiasis are not more frequently encountered in children in view of the accepted importance of infection in the etiology for adults and

the prevalence of focal and general infections during childhood. Intestinal parasites, also predominating in childhood, are reported in some instances as forming the nuclei of gall stones.

The radiologist apparently has not been called upon, or perhaps has failed to be of assistance in the diagnosis of gall bladder disease in children. Cholecystography, by Graham's method, has been practised almost exclusively with adults

Experience is needed with this method in children, first better to establish the normal and later to assist in the diagnosis of disease.

The feasibility of making an efficient x-ray examination of a wriggling, crying child is well known to radiologists. The development of technical facilities allowing a hundred or more milliamperes for less than 1/10 second exposure at very low penetration overcomes the blurring effect due to respiration and other movement. We should be able to show a higher percentage of gall stones in children than in adults. Our equipment for work with children is not, however, generally known to pediatricians and others who see these occasional children with gall stones and cholecystitis. When the condition is borne in mind, and it is understood that the radiologist is able to assist in the diagnosis, I believe more cases will be recognized before the operation or the autopsy.

CONCLUSIONS

A review of the literature and the increasing number of cases reported indicate that cholecystitis and cholelithiasis in children is of more frequent occurrence than is commonly believed.

The conditions are not unusually difficult of diagnosis if their occurrence is borne in mind.

The radiographic demonstration of gall stones in a child, age three years, which forms the basis for this report, lends encouragement to the belief that radiologic methods should more often be of assistance in arriving at the correct diagnosis.

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jaundice was noted. The author regarded these cases as due to functional disturbance of the gall bladder of a spastic nature and treated them medically.

42. Von Khautz, A.: Cholelithiasis und Cholecystitis im Kindesalter. Centralbl. f. d. Grenzgeb. d. Med. u. Chir., 16:545, 1913. Note: Reports no cases; a review of literature.

43. Mathews, F. S.: Gall Stones in the Young. Ann. Surg., 59:781, 1914. Note: Mathews' cases were in patients sixteen and twenty-one years of age, respectively. See Downes.

44. Eustermann, G. B.: Disease of the Gall Bladder in the Young. Ann. Clin. Med., 2:283, 1923-1924. Note: A general review of 117 cases of gall bladder disease in children and young adults; no details of cases given; mentions occurrence of gall stones in young patients with congenital hemolytic icterus.

DISCUSSION (*Abstract*)

Dr. Charles J. Bloom, New Orleans, La.—Although the subject of gall bladder disease in infancy and childhood had been described as far back as 1722 by Gibson, so far as I was able to find out there have been reported less than 150 cases. Gall bladder disease in infancy and childhood is uncommon, particularly in infancy. In older children, especially those from six years up to puberty, we meet with a condition that often suggests the possibility of cholelithiasis, namely, there is a large group of children who in the beginning have been fed on a high carbohydrate diet, condensed milk in infancy, excessive amounts of cereals, sugar, candies, sugar cane or molasses. This type sooner or later develops a condition which we accept as chronic intestinal indigestion, and a large percentage of this group at some time or other has definite signs of inflammatory lesions of the gall bladder and jaundice to varying degrees. We have had cases that vomited for days, with associated abdominal pain and an icteric appearance, and still we have been unable to prove that there was any association between these cases and gall stones. In my service at the Charity Hospital we have something like fifty beds. We have a great many postmortems. I do not know a single case that has been reported as gall stones. However, seeing the pictures and reviewing the literature might prompt us to consider this in a different light. Perhaps in the future we shall be able to add to the collected cases some which we may have overlooked.

Dr. Evarts A. Graham, St. Louis, Mo.—Increased interest in gall bladder disease in infancy and childhood has been partly due to better diagnosis of gall bladder disease now-a-days than formerly.

There is a group of cases in early infancy with concretions in the gall bladder discovered at post-mortem examination which are really nothing but bile pigment; they are not true gall stones. It is doubtful that they cause trouble later on. They are somewhat similar to the uric acid infarcts which one sees in the kidneys of new born infants at times, also probably without much significance. There are also sometimes found in the biliary tract of new born infants obliterations of the bile passages which cannot be considered as gall bladder disease in the ordinary sense. There are other cases in which there is unquestionably gall bladder disease clinically demonstrated, and demonstrated also by x-ray and by pathologic examination either at operation or at autopsy. It is these cases that are of extreme interest and it is to this group that Dr. Beals's case belongs.

Dr. Bloom, in his discussion, mentioned the fact that he had never seen a case of cholecystitis in early childhood, but he spoke of cases of gastro-intestinal disturbances with jaundice which he attributed to dietary indiscretion. I am not a pediatrician and therefore I cannot argue out this subject to its finality with him, but I can say that the prominent symptom of gall bladder disease in adult life is indigestion and dyspepsia. That is the chief symptom present in over 80 per cent of the cases of gall bladder disease. Furthermore, many cases of jaundice in childhood and infancy also pass under the term of catarrhal icterus. Many of the same cases which in adult life do not receive this obscure title but receive the more definite title of biliary tract disease. Catarrhal icterus is not an entity at all; it is merely a clinical syndrome in which the chief symptoms are icterus and gastro-intestinal disturbances. There is no pathologic entity to be found known as catarrhal icterus. Virchow, who was the first to give it this name, thought it was due to blocking of the common duct with a plug of mucus, because in the post-mortems on patients who died of so-called catarrhal icterus, he found the common duct plugged with mucus, and that is why the name catarrhal icterus is given to this condition. As a matter of fact, any inflammation of the liver, whether associated with gall bladder disease, or whether merely due to gastro-intestinal inflammation, may give rise to this clinical syndrome which is often spoken of as catarrhal icterus. Since the development of cholecystography we have been paying more attention to cases in the St. Louis Children's Hospital with gastro-intestinal disturbances, some with icterus, some without icterus; and we have found many cases, some of which have been operated upon, some with stones and some with thickened adhesions in children under ten years of age. I cannot tell you how many cases, as I did not come prepared to discuss this paper, but I can recall some five or six cases in the last year or two in children under ten years of age which have been confirmed by operation. In addition to these there have been a considerable number of other cases in which we have made a diagnosis of chronic cholecystitis, but which for various reasons were not submitted to operation. If our clinical and radiological diagnoses have been correct it will bring the number to somewhere between ten and fifteen. Some of these cases have been in infancy. One patient was two years old. This case was not confirmed by operation, however, so we are not sure; but several cases were under fire. I believe, if I may express an opinion, that cholecystitis is not nearly so rare in childhood as we ordinarily think it is, but it is not so common in childhood as it is in adult life. It takes many years for the so-called clinical picture of chronic cholecystitis to develop, and therefore we cannot expect to see it commonly in childhood. The child has not had the time to develop the picture. I believe if the pediatricians think of gastro-intestinal disturbances and catarrhal icterus more in terms of chronic cholecystitis that more cases of gall bladder disease will be recognized.

CORRELATION IN TEACHING DURING PRECLINICAL TERMS*†

By J. T. HALSEY, M.D.,
New Orleans, La.

Retrospection and comparison often serve as a useful starting point in an effort to form opinions, make criticisms and offer suggestions. As, aside from myself, few here present remember the conditions usually obtaining even in the better medical faculties in this Country during the last decade of the Nineteenth Century, I may be pardoned for referring to certain of them. In those days, with rare exceptions, the teachers of the fundamental sciences were practicing physicians or surgeons, whose knowledge of the sciences, for the teaching of which they were responsible, might be either reasonably sufficient or relatively superficial. However they were usually men of ability, conscientious and well versed and interested in clinical medicine and surgery. Consequently, while at that time one heard nothing of correlation in medical teaching, I believe my contemporaries will agree with my recollection that many at least of these fundamental science teachers laid sufficient, perhaps too much, emphasis on the significance and practical bearing of various phases of these subjects. As most of the clinical teachers had little knowledge of and less interest in the preclinical sciences (except that the internist was usually well up in pathology and the surgeon equally so in anatomy), the clinical significance of physiology, biochemistry or pharmacology was seldom if ever mentioned in the clinical teaching.

Sometime later while the conditions in the clinical departments remained much the same, there was a marked change, and for the better, in the preclinical departments. The heads of these were now as a rule all-time men, who not only had graduated in medicine but who had also sufficiently pursued the special study of their chosen subjects. Although there were exceptions to the general rule, these men, while essentially and primarily anatomists, physiologists, and so forth, by virtue of their earlier study of medicine, had some knoweldge at least of clinical subjects and of the future tasks and problems of their students. Consequently in their teaching these were not forgotten and during this period there was some correlation in teaching in the preclinical years.

Today the picture has changed or is rapidly changing. In more and more of our schools a constantly growing proportion of the clinical teachers have not only a good training but also a live interest in the fundamental medical sciences. Consequently in their teaching reference is often made to the necessity for and value of a knowledge of theories and facts which have been learned (and too often forgotten) in the earlier stages of the curriculum. That there is yet enough of this sort of correlation in the clinical teaching I do not believe, but a good start has been made and I hope and believe that this tendency will continue to grow. Unfortunately (as I think) an opposite change has occurred in the preclinical departments. Here we find that a rapidly growing percentage of heads of departments and subordinates are individuals who have not studied medicine and who consequently have little or no knowledge of the problems and tasks of the practitioner. Many of these teachers confess, while some boast, that they are not interested in the clinical significance of different phases or portions of their subjects. Is it not to be expected that courses planned and conducted with such a point of view will often fail to arouse an interest in and enthusiasm for the subject in young men whose aim is to become competent physicians and surgeons? Is it not probable that courses in physiology, biochemistry, anatomy or pharmacology conducted by these teachers will all too often be characterized by undue emphasis on phases of the sciences especially interesting to the teacher but which today are of little significance in medicine, and that they will fail adequately to emphasize and teach parts of the subject of especially great importance and interest for the future physician? While I could cite by name more than one department not presided over by graduates in medicine, in which the courses have been preeminently valuable to medical students, I believe that the doubts expressed above are justified by conditions all too prevalent today.

If they are justified what is the remedy? Shall we insist on the M. D. as a prerequisite for teachers in the preclinical branches? Even were it possible to do so (but it isn't), I would unqualifiedly say, no. To my mind the prevailing defects (and I believe they are real ones) can be overcome if they are realized and the necessary effort made. Teachers of the preclinical sciences, be they Ph.Ds. or M. Ds. should seek and cultivate close association with and

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*Read in Section on Medical Education, Southern Medical Association, Twenty-First Annual Meeting, Memphis, Tennessee, November 14-17, 1927.

†From the Medical Department Tulane University of Louisiana.

both advice and aid from their clinical colleagues, many of whom are sufficiently trained and interested in the fundamental branches to be of real service. In addition I believe that the head of each and every one of the preclinical divisions must have the active assistance of clinicians if students are to profit to the full from these courses. This request of the preclinical teacher for assistance from the Clinicians should not be confined, as it usually is to the request, that the clinicians should as often as possible show to their classes, the clinical bearing and importance of facts or theories from the clinical sciences nor to a demand that during the clinical years place be made for additional instruction in his branch, valuable as such course might be.

He should invite the clinicians from time to time to present to his class actual patients, for the understanding and appreciation of whose conditions, a knowledge of certain facts or theories of the preclinical science is essential.

To anatomical students, the surgeon may show, with x-ray pictures, fracture or dislocation cases, pointing out the influence of muscles and tendons in causing displacement or deformity; or the neurologist, using plates or lantern slides of normal and pathological cord sections, may show patients with different cord lesions and correlate anatomical lesion with resultant disturbance of function. In students in biochemistry and physiology, the demonstration, with history, laboratory findings, and records, of cases of diabetes, thyroid disturbance, gout, nephritis, dyspnea, cardiac arrhythmias, and soforth, could not fail to increase interest in and enthusiasm for the study of these preclinical subjects and should spur the students on to a greater effort. Moreover such clinics should aid in clarifying and fixing more firmly the student's knowledge and understanding of scientific fact and theory. Such examples should be sufficient to indicate to you why I believe that occasional clinics, showing to students in the preclinical terms a moderate number of carefully chosen and skillfully presented patients, would be useful in the teaching of the preclinical sciences.

By and large the medical student of today is much more fortunate than those of forty, twenty or ten years ago. His opportunities to become qualified as competent physician or surgeon are immeasurably greater than of yore. On the other hand, the task with which he is confronted is a formidable one, beyond the powers of accomplishment for many a matriculate, even though his road be made as easy and his burden as light as it is proper and possible to make it. The first two years are especially difficult for him. For all but the exceptionally gifted the price of success is unremitting labor. Many weary of this, because they cannot see the use and need of much which they are called upon to learn. Therefore interest fails and, where interest is lacking, learning is more difficult and effort tends to lag. All students possessing the qualities which a good physician will need, are not necessarily imbued with a thirst for knowledge for its own sake, nor are they always capable of growing enthusiastic over the wonders of an endothelial cell, the action current of a nerve, the sympathomimetic action of a drug, formula of xanthin bodies, or the contortions of an ameba. A student may not have this type of intellect and yet have the mental ability and the type of character and personality even more needed in his chosen profession. All too large proportion of medical students find the work in the first two years not only extremely difficult but also most uninteresting. Were it made more interesting it would be less difficult. I believe that such clinics as have been outlined above, by supplying the lacking interest, would produce on students of the first and second years an effect comparable to that produced on foot-sore and exhausted soldiers by the music of a band. That such clinics would thus not only lighten the task but also increase the student's ability to work and improve his achievements is my firm conviction.

Thus far only correlation between the preclinical and clinical branches has been discussed. But correlation should not stop here. It should also obtain among the preclinical divisions. In the teaching of one of these, pharmacology, there is of necessity a close correlation with physiology and I believe that this is universally the case in this branch. However I believe it of great importance that in teaching pharmacology the teacher should bear in mind not only correlation with the department of physiology but also with those of biochemistry, anatomy, bacteriology, and pathology. I realize that, owing to its coming later in the curriculum than most of the other preclinical subjects, correlation is easier for the pharmacologist to carry out and that the necessity and advantage are here more apparent than elsewhere. In spite of these advantages it is my impression that relatively seldom is there sufficient attention paid to correlating this course with any of the preclinical subjects, and particularly that the bearing of pathology and bacteriology and of pharmacology on each other is seldom fully realized or sufficiently stressed. Partly because it is much more diffi-

cult but also because its value is not sufficiently realized, I believe that all too often anatomy and biochemistry departments fail even to attempt to correlate with the other preclinical branches and that most teachers of physiology and of pathology and bacteriology fail sufficiently to appreciate the value of and opportunities for correlating their teaching with that of the other preclinical subjects.

If this is true, and I claim that it is, the remedy is a closer association between all teachers in the preclinical divisions, permitting of interchange of ideas, discussion of problems, both pedagogic and investigative and seeking for and, when asked to do so, giving advice and assistance to colleagues in the different departments. Combination seminars, journal clubs and faculty lunch table, especially the latter, should aid in attaining these objects. However the prime essential to obtaining such correlation is that the preclinical teachers should realize the value, nay, the necessity thereof.

Criticism, especially from the preclinical side, of such views has often been made to the effect that it is the function of departments to teach their own subjects and that these are so extensive that time does not permit doing even this without attempting to aid other divisions. To this my answer is that, under present conditions, it is impossible to help medical students to acquire a reasonable knowledge of more than those portions of any subject, which are of fundamental significance and in addition those of especial importance to the future physician and that, by concentration or even omission of other portions, room can be made for the important correlative teaching. Another criticism is that paying especial attention to portions of a subject which have clinical significance, tends to introduce a utilitarian spirit and to impair the scientific attitude of the student. Personally I have never seen why a careful, reasonably complete discussion, in the proper so-called scientific spirit, of a chapter which is of importance to the physician and is both valuable and interesting to the medical student should be less worth while than one devoted to subject matter lacking these attributes. Interest in and zeal for logical exact thinking can be just as well, I believe, induced by the right kind of teaching of so-called practical things as by the teaching of things whose practical significance is as yet not apparent.

DISCUSSION (*Abstract*)

Dr. J. H. Musser, New Orleans, La.—I agree with Dr. Halsey upon the necessity, to a certain extent at least, of correlative courses but I think it must be borne in mind that everything is not quite so rosy as Dr. Halsey would paint it. If one has to teach a student always the reason why he is learning something and does not give him an opportunity of using his powers of reasoning, in time it will result in a certain amount of mental apathy in those students. In courses in the university other than medicine, in engineering for example, it is not necessary to explain to a student why certain subjects are required, and where the practical applicability comes in.

The correlative courses are, in a measure, of value in the last two years. If a student, after he has learned in his first two years the basic principles, can appreciate the why and the wherefore of what he is dealing with, then he can reason and he can be shown why certain symptoms occur and why certain physical signs are manifested in a particular disease. If students know the pathology and disturbed physiology of certain disorders there will be no necessity of their sitting down and memorizing a whole list of symptoms and signs. They will have the basic information and can use it. Whether it is necessary to teach them why they are learning that a certain muscle does this or that is a question in my mind.

Dr. H. T. Marshall, University, Va.—One value of such a curriculum as Dr. Halsey describes is its adaptability to changing needs. We all know the difficulties of finding a place in our inelastic, overcrowded schedule for new subjects. We must figuratively split the curriculum with a wedge in order to find a place in which to squeeze awkwardly a new subject. It is going to be increasingly difficult to keep the present type of curriculum in tune with advances in knowledge. Under the scheme I have suggested there will be little difficulty on this score. Since each of the dozen major subjects will be taught as a rounded entity, rearrangements or changes in emphasis will be easy to make as the prospective alters.

Southern Medical Journal
JOURNAL OF THE
SOUTHERN MEDICAL ASSOCIATION

Published monthly by the Southern Medical Association, Empire Building, Birmingham, Ala. Annual subscription $4.00. Single copies 35c each.

Entered as second-class matter at the Post Office at Birmingham, Ala., under Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1106, Act of October 3, 1917, authorized December 20, 1921.

DR. M. Y. DABNEY, Editor
C. P. LORANZ, Secretary-Manager

Vol. XXI AUGUST 1928 No. 8

EDITORIAL DEPARTMENT

SOUTHERN MEDICAL ASSOCIATION
Twenty-Second Annual Meeting
Asheville, North Carolina
November 12-15, 1928

CARCINOMA OF THE RECTUM

With the increasing number of rectal specialists in the South, rectal cancer should be recognized earlier and treated more thoroughly in this territory. The origin of this type of malignant growth and its early stages are described in a recent paper of Lockhart-Mummery and Dukes, of London.[1] Among the causes of the condition, they believe chronic pruritus ani and chronic hemorrhoids may rarely be classed; and there is no evidence that chronic constipation is an exciting cause. Adenoma, they believe, is the only condition in the rectum which predisposes to cancer with any frequency.

For many organs, chronic hyperplastic changes such as those which Sir Lenthal Cheatte describes in the mamma, have been recognized as commonly preceding cancer. Similar epithelial proliferations frequently precede cancer of the rectum, according to the above authors.

Adenomata of the mucous membrane of the large intenstine are peculiarly liable to undergo malignant change. In the rare cases of muliple adenomata, an inherited condition, malignant change always occurs in one or more of the tumors. The usual simple adenomata may show both benign and malignant sections.

If one removes a piece of bowel for cancer of the rectum, irons out and examines its walls with the microscope, one may see irregularities of the wall due to epithelial hyperplasia. This is found most often in association with multiple adenomata and cancer of the rectum, more frequently near a small malignant tumor than a large malignant ulcer, and it affects an extensive area of bowel several inches above and below the cancer. The adenoma represents only a more advanced stage of hyperplasia.

In the development of cancer one finds: first, localized patches of hyperplasia invisible to the naked eye, discoverable with the microscope, affecting an extensive area of bowel; the second stage is the appearance of a crop of sessile adenomata over as wide an area as the initial hyperplasia; third, cancer appears either in one of the pre-existing adenomata or in the neighboring epithelium; and fourth, there is progressive enlargement of the tumor, and retrogression of the hyperplastic changes and benign surrounding tumors.

Adenomata tend to recur after removal, not in the same place but as the result of a progressive hyperplastic change in a fairly extensive area of bowel epithelium. Hence, the patient with multiple adenomata should be observed at frequent intervals.

The earliest recognizable lesion in cancer of the rectum, according to the London authors, is a hyperplastic change taking the form of irregular proliferations scattered over an extended area of bowel. Following this is the adenoma; and carcinoma formation is "an accident happening to a previously existing adenoma."

Rankin and Broders,[2] of the Mayo Clinic, discussing prognosis in cancer of the rectum, agree that the type of neoplasm found higher in the rectum, away from the anal orifice, is practically always adenocarcinomatous. They have compared the degree of malignancy, classified according to Broders' method by the microscopical findings, and its relation to prognosis. They find that if carcinoma of the rectum is graded by Broders' method 1, 2, 3, or 4, the average duration of life decreases as the grade increases.

1. Lockhart-Mummery, J. P.; and Dukes, Cuthbert: The Precancerous Changes in the Rectum and Colon. Surg. Gyn. Obst., 46:591, May, 1928.

2. Rankin, F. W.; and Broders, A. C.: Factors Influencing Prognosis in Carcinoma of the Rectum. Surg. Gyn. Obst. 46:660, May 1928.

Carcinoma of the rectum, as with carcinoma elsewhere inside the body, is apt to produce few symptoms in the early stages. It is a question whether the yearly complete physical examination which physicians are recommending for normal persons, should include gynecological, urological and rectal investigations, as well as a variety of laboratory tests.

MEASLES PROPHYLAXIS

Starting with the prevention of smallpox by vaccination with the virus of cowpox, the list of preventable contageous diseases has steadily increased. With the advent of bacterology as a science at the end of the Nineteenth Century, and the isolation of specific organisms for a large number of diseases, the prospect seemed fair and a method at hand for the complete eradication of diseases due to micro-organisms.

The typhoids are now prevented by vaccine; yellow fever and malaria, by eradication of the mosquito pests which bear them; diphtheria and tetanus, by administration of antitoxin; and even some progress is claimed in France in the control of tuberculosis by vaccination with *Bacillus Calmette Guerin*. In the last epidemic of infantile paralysis, convalescent serum was used to advantage as a preventive measure.

Measles has been called the most contageous of all the contageous diseases, since practically the entire population is susceptible. It has been considered of no great importance, one of the necessary evils in childhood, and has received no great amount of attention. In pediatric text books, methods of prevention are not usually dwelt upon, and treatment is generally described as "symptomatic," yet measures for measles prevention have been described and have been fairly extensively investigated.

Prevention is apparently accomplished by giving to the exposed patient serum obtained from a measles convalescent soon after defervescence; or by giving a much larger quantity of serum from an adult who has had measles in childhood.

Nicolle and Conseil first used convalescent serum in immunization against measles in 1916. In 1923, Von Torday summarized two thousand reported cases and found only 3 per cent not completely immunized. Degkwitz, working in Pfaundler's Clinic in Munich, used adult blood, since convalescent blood is difficult to obtain, while adult blood is practically always available.[1] [2] [3]

Complete passive immunity from the serum lasts only two to four weeks. If a small amount of serum is given after a known exposure a very mild attack of the disease occurs, which results in a permanent active immunity.

The serum should be given within the first four days following exposure. Degkwitz advises 5 c. c. of convalescent serum intravenously within four days of exposure in order to give complete protection to a child under three years of age. Two to three cubic centimeters of convalescent serum are said to give a modified attack to the same child; and 15-20 c. c. of adult serum will frequently result in a modified attack of measles when given within four days of exposure. A number of favorable reports have appeared on measles prevention.[4] [5]

Tunnicliff has isolated a green-staining diplococcus as the supposed etiological agent in measles. Others have isolated supposed measles organisms, but this one has apparently best held its ground. She has inoculated goats with it and has used immune goat serum just as the convalescent serum is used. Good results were reported with it two years ago.[6] [7] Hoyne and Peacock[4] have recently reported another satisfactory series of cases in which modified measles were obtained by injecting the goat serum five days or more after exposure. The immune goat serum has not yet been put on the market.

Measles, on account of its frequent complications is deserving of serious consideration. For protection of young children in a family where one case has developed, serum prophylaxis is

1. Karelitz, Samuel and Devin, Samuel: Measles Prophylaxis. Amer. Jour. Dis. Ch. 33:408, March 1927.
2. Bivings, F. L.: Measles Prophylaxis of the Use of Parents Blood Serum. Preliminary Report. S. M. J., 20:735, Sept. 1927.
3. Bivings, F. K. and Dickson, R. W.: Parental Blood Serum in Measles Control. Arch. Ped. 44:629, Oct. 1927.
4. Hoyne, A. L. and Peacock, S.: Prevention and Modification of Measles with Antimeasles Diplococcus Goat Serum. Amer. Jour. Dis. Ch. 35:1021, June 1928.
5. Toomey, J. A.: Prophylaxis of Measles. Amer. Jour. Dis. Ch. 32:401, Sept. 1926.
6. Hoyne, A. L., and Gasul, B.: Measles Prophylaxis. Report of Use of Immune Goat Serum. J. A. M. A. 87:1185, 1926.
7. Tunnicliff, Ruth and Hoyne, A. L.: Prevention of Measles by Immune Goat Serum. J. A. M. A. 87: 2139, Oct. 9, 1926.
8. Forbes, R. P. and Green, B.: Modified Measles. J. A. M. A. 89:1601, 1927.
9. Kingsbury, A. N.: Serum Prophylaxis in Measles. Lancet, 1:7-8, Jan. 1, 1927.

valuable. Measles as a regularly recurring scourge in children's hospitals and in other child-caring institutions, should always be treated by prophylactic serological measures.

PARATHYROIDS IN FRACTURE HEALING

It has been accepted that fractures do not heal if the blood calcium is low, as it frequently is in rickets, and in certain other conditions. The quantity of calcium in the circulating blood is to a large extent controlled by the parathyroid glands. It has been shown that an acid extract of parathyroids of one animal has pronounced constant effects upon other species, when administered orally or in the vein. Parathyroid extract raises the blood calcium, leading if administered too long, to hypercalcemia, coma, and death. If all the parathyroids are extirpated tetany, convulsions, low blood calcium and death ensue.[1] Since blood calcium and parathyroid secretion are closely related, and fractures do not heal in the presence of low blood calcium, logically the condition of the parathyroids should influence fracture healing.

The relation of the parathyroids to healing of fractures remains to be shown. Some of the effects of parathyroid extirpation on bone union have been indicated by Ross[2], of McGill University, working with cats, and making periodic x-ray examinations of the fractures. The removal of two of the parathyroids did not delay bony union or affect the blood calcium. The removal of as much as three parathyroids delayed union frequently four to five weeks, and caused a drop in the blood calcium of 2 to 3 mg. per 100 c. c. of serum. After a period the calcium level rose to normal, and bony union occurred.

Complete immobilization of the limbs was not maintained for more than forty-eight hours. Callus appeared in fifteen to sixteen days in the normal animal, and union was firm in twenty-one days. In the first five to six days following fracture in normal cases, there was invasion of the blood clot formed at the site of fracture, by connective tissue. In ten to eleven days, the blood clot was gone, and was replaced by connective tissue, probably a small amount of cartilage, and here and there small areas of new bone. In the cats from whom three parathyroids were removed, the process of repair from there on was delayed.

These studies serve further to emphasize the importance of non-mechanical factors in fracture healing. Perfect carpentry is invaluable in setting human fractures, but adequate repair material in the blood itself is also essential. Knowledge of the normal constituents of blood is very useful here or in various other fields.

1. Editorial, Parathyroid Harmone, JOURNAL, 21:3, March 1928, p. 245.
2. Ross, D. E.: Relation of Parathyroids to the Healing of Fractures, as Controlled by Roentgen Rays. Arch. Surg. 16:922, April 1928.

Asheville—Where We Meet

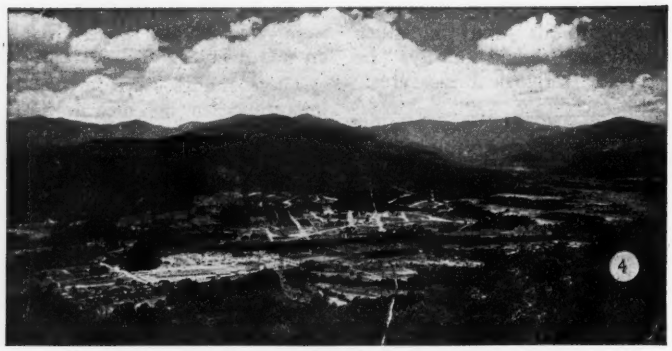
ATTRACTIONS OF ASHEVILLE*

Members of the Southern Medical Association, when they gather for the annual convention in Asheville in November will find a hospitable welcome and a hearty cooperation extended to them by the citizens of this mountain resort city. Every possible facility to further the success of the convention will be offered. The Buncombe County Medical Association is bending every effort to the task of preparing entertainment for visitors and providing proper convention space and space for exhibits.

Statistics in regard to climate show that the month chosen is one of the most delightful of the year in the Land of the Sky. Yearly averages show that in November fifteen days of the month are clear, six are partly cloudly and only nine days on the average are cloudy. This is the time of year also when the mountainsides will be colored with the glorious hues of the changing leaves. The autumn radiance begins at the summits and advances down the mountain slopes during the exhilarating Indian Summer days of late October and November.

The climate of the Land of the Sky has long been noted for its excellence. Owing to a high altitude (2,300 feet at Asheville), the atmosphere is unusually dry. The altitude is also conducive to cool summer weather, the average temperature for the summer months being seventy-two degrees. The protecting barrier of mountains which surround the plateau on which Asheville is situated prevents any extreme changes of temperature during the winter months and wards off the worst winter storms. The average winter temperature is thirty-nine degrees. The golf courses in and near Ashe-

*By John D. Topping, Publicity Director, Asheville Chamber of Commerce.



Bee Tree Watershed, the source of Asheville's municipal water supply.

ville are open for play throughout the winter months.

Asheville's five general hospitals may be of interest to many of the delegates. The Mission Hospital is an institution which receives its support from public funds. It is one of the largest and best equipped hospitals of the city. The Biltmore Hospital, the Meriwether Hospital, the French Broad Hospital and the Blue Ridge Hospital are units of the general hospital equipment. The last named institution has been established to care for the colored patients.

Asheville's water supply, coming from the slopes of the highest mountain of this region is of high quality and purity. Western North Carolina mountains in their highest summits are composed of granite or other insoluable material and hence the water which reaches the city mains at Asheville is practically as free from solids as is distilled water. Many automobile owners in this region habitually use water from the city mains in their automobile batteries.

Owing to the excellent climate and the pure surface waters of this region as well as the magnificent mountain scenery, Western North Carolina has been chosen as the site for nearly one hundred camps for boys and girls. The camps operate throughout the summer and early autumn months and draw to Western North Carolina hundreds of young campers from all parts of the Eastern United States.

Several years ago Asheville undertook a community smoke abatement campaign which has been very successful in its results. Through the cooperation of the Southern Railway, the large industries, the laundries, hotels and other large users of fuel, the atmosphere has been greatly clarified. All of the more important industries and institutions have installed special smoke-consuming equipment in their furnaces and owners of residences in the metropolitan area use, for the most part, a smokeless coal.

Stringent health regulations have made Asheville one of the most healthful cities in the United States. Growth in educational facilities and in commerce has been rapid here in recent years. The city today is a well-rounded, modern metropolitan municipality.

Book Reviews

The Surgical Treatment of Malignant Disease. By Sir Holburt J. Waring, M.S., M.B., B.Sc. (London), F.R.C.S., Surgeon to and Joint-Lecturer in Surgery at St. Bartholomew's Hospital and Medical College; Consulting Surgeon to the Metropolitan Hospital; Examiner in Surgery, University of Oxford; Late Vice-Chancellor and Dean of Faculty of Medicine, University of London; Late Member of Court of Examiners, and Vice-President, Royal College of Surgeons of England; Late Examiner in Surgery, Universities of London and Manchester; Past President of Medical Society of London. 667 pages illustrated with 19 color plates and 277 figures. New York and London: Oxford University Press, 1928. Cloth, $15.00.

This book represents the liberal expansion of a Bradshaw Lecture, given by the author in 1921. The subject in the main, has been treated from a clinical and surgical point of view; but to a limited extent, the pathological aspect of malignant disease, in so far as it has bearing on clinical diagnosis or surgical treatment, has been dwelt upon. A chapter is devoted to each organ or separate anatomical area; the malignant disease of this area is discussed systematically with regard to incidence, pathology, symptoms, clinical appearance, diagnosis, treatment, prognosis and results. As proof of his conscientious treatment of the subject, individual chapters are included dealing with the occasional tumors of such neoplas-

tically spared organs as ear, fallopian tube, scrotum, urethra and umbilicus. He has introduced semi-diagrammatic illustrations of the lymph vessels and lymph glands in connection with each organ or anatomical area under consideration.

Unfortunately the follow-up clinics of St. Bartholomew's Hospital have not contributed greatly to the statistical evidence bearing on the results of the surgical management of malignant disease. Lord Waring quotes extensively from an article by Balfour of the Mayo Clinic, published in 1916, when the Percy cautery was still used in the treatment of advanced inoperable cervical cancers. On page 18, in reference to the preoperative use of x-ray and radium, he definitely states: "In some cases the rate of growth of the neoplasm is increased." This latter statement is in contradiction to the opinions of most experimental pathologists and radiologists.

As a whole the book is a model of its kind. Printed in large type, profusely and pertinently illustrated, replete with carefully selected bibliographic references, complete with details of surgical procedures, reflecting a wealth of personal experience, and charming in its style of presentation, it was such a type of book that Channing had in mind when he said: "In the best books, great men talk to us."

A Book for Us Diabetics and Our Doctors. By Don H. Duffy, M.D., 80 pages. Illustrated. Published by the Author at South Lancaster, Mass. $1.50.

The problem of educating the diabetic patient is one that is constantly confronting the internist. Unfortunately, the mental and educational equipment of all diabetics will not permit them to undertake a completely comprehensible study of the disease that will usually be with them for the remainder of their days. In spite of this fact, most of the so-called simple books for the diabetic patient are entirely too technical. This short book is the essence of simplicity. It is entirely practical and patients should have no trouble in understanding it. It is written in almost a conversational style and difficult medical terms are avoided. The author does not attempt to tell the patient exactly how much insulin he requires or how many grams of carbohydrate, protein and fat he should consume. He leaves the actual management to the physician, as should be. He does make, however, many excellent suggestions as to food values and of food substitutions which will not only be helpful to the housewife in planning palatable menus but also to the physician in the management of the diet. After reading it, the patient becames easily initiated into the mysteries of his disease and its treatment and his attitude will be one of understanding and confidence rather than distraction and fear.

The New York Academy of Medicine Lectures on Medicine and Surgery. First Series 1927. 319 pages with 39 illustrations. New York: Paul B. Hoeber, 1928. Cloth, $5.00.

This collection of lectures, of a practical nature which were given at the New York Academy of Medicine, is worth while. It is made up of fifteen papers by men who are known to be well qualified in their chosen subjects. These subjects are wisely selected. The material is intended for the general practitioner and is therefore diversified. Medical and surgical problms are considered in a practical way and there is not a single article that is not well worth reading. It would be impossible to mention them all, but MacKee's paper on the "Cutaneous Manifestations of Syphilis" is outstanding. Reisman has an excellent article on the "Treatment of Pneumonia." Poole's "Surgical Aspects of Diseases of the Thyroid" and Erdman's discussion of "Intestinal Obstruction" are both excellent.

Symptom Diagnosis, Regional and General. By Wilfred M. Barton, A.M., M.D., F.A.C.P., Associate Professor of Medicine, Medical Department of Georgetown University; Attending Physician, Georgetown University Hospital; Medical Consultant, Columbia Hospital for Women; Chief of Medical Service, Gallinger Memorial Hospital, Washington, D. C., and Wallace M. Yater, A.B., M.D., Fellow in Medicine, Mayo Foundation, Rochester; Formerly Professor of Physical Diagnosis, Medical Department of Georgetown University; Associate Attending Physician, Garfield Memorial Hospital, Washington, D. C. 851 pages. New York: D. Appleton and Co.

This book fulfills its purpose in a very efficient way. The authors state that this purpose is: first, to aid in making a diagnosis; second, to prevent the oversight of important conditions; and third, to make the physician more observant of the characteristics of symptoms. It is, in reality, a very complete catalogue of symptoms, giving the various possibilities of diseases of which they may be manifestations.

The arrangement of the contents is well worked out and eliminates lost motion in reference. Regional symptoms are first discussed and make up the major part of the book. Symptoms referable to various parts of the body are taken up according to their topographical location and many sub-references to other parts of the book are given to avoid useless repetition. General symptoms are considered in the last part of the book. The chief weakness is that the index is of symptoms and does not list diseases.

A Text-Book of Actinotherapy with Special Reference to Ultra-Violet Radiation. By D. D. Rosewarne, M.R.C.S. (Eng.), L.R.C.P. (Lond.), Late Honorary Actino-Therapeutist and Assistant Physician, City of London and East London Dispensary; Specialist Pathologist, R.A.M.S.; Officer in Charge, Central Military Laboratory, Port Said; Officer in Charge, Anzac Field Laboratory, Sinai, Palestine; Late Assistant Pathologist and House Physician, St. Mary's Hospital. 237 pages, illustrated. St. Louis: C. V. Mosby Company. Cloth, $4.00.

Methods of procedure are carefully explained and the physics of actinotheraphy is well presented considering the elementary character of the book.

Diseases of the Mouth. By Sterling V. Mead, D.D.S., Professor of Oral Surgery and Diseases of the Mouth, Georgetown Dental School; Professor of Diseases of the Mouth, Georgetown Medical School; Oral Surgeon to Georgetown Hospital, Washington, D. C., 578 pages with 274 illustrations. St. Louis: C. V. Mosby Company. Cloth, $10.00.

This is a terse encyclopedia of oral diseases. All diagnosticians should read that part of the chapter on pulpless teeth which refers to "when to extract."

The subject matter is descriptive, yet not voluminous. Generously illustrated, it is a good textbook and a wonderful reference book.

Southern Medical News

DR. WILLIAM LeROY DUNN, VICE-PRESIDENT—IN MEMORIAM

The Southern Medical Association is fortunate in never having lost by death a president in office; and until quite recently no vice - president has ever died in office. However, on May 24, 1928, the Association suffered a severe blow in the death of its Second Vice - President, Dr. William LeRoy Dunn, of Asheville, North Carolina.

Dr. Dunn was born in Fairfield, Ohio, in 1871 and later moved to Decatur, Ohio, where he was graduated from high school with the record of having been the youngest student ever to have completed the course of study.

His college work was done at the University of Michigan, where in 1889 he took the degree of Pharmaceutical Chemist; Doctor of Medicine in 1891; Bachelor of Science in 1894; and in 1916 his Alma Mater conferred an honorary Master of Arts upon him in recognition of his splendid work in the study of tuberculosis. It is of interest to note that Dr. Dunn was graduated in medicine at the age of twenty and had to wait a year before he could secure his license to practice.

In 1894, Dr. Dunn came to Asheville because he had contracted pulmonary tuberculosis, and lived not only to be cured of it but to become one of the national authorities in the diagnosis and treatment of that disease. On reaching Asheville he was privileged to work at Winyah Sanatorium with the distinguished Swiss physician, Dr. Arnold C. Klebs, whose name is linked with that of Loeffler in connection with the discovery of the diphtheria bacillus.

An internist in Asheville, Dr. Dunn naturally became deeply interested in the study of tuberculosis clinically as well as chemically, and contributed various important papers on the subject.

During the World War he was commissioned Major, later was made Lieutenant-Colonel and after the War was made Colonel in the Reserve Corps. He was also appointed Colonel by the Italian Army and held a commission as Colonel on the staff of the Governor of Kentucky. Dr. Dunn saw service in Italy as Chief of the Medical Service of Base Hospital No. 102, at Vicinza. However, he was made American Consultant of the Expeditionary Forces in Italy and traveled extensively over Italy in that capacity.

Dr. Dunn was a Vice-President of the North Carolina State Medical Association; a member of State, National, International Tuberculosis Associations; was only recently elected President of the American Climatological and Clinical Association; and at the Memphis meeting of the Southern Medical Association was elected Second Vice-President.

By the irony of fate Dr. Dunn died of carcinoma of the lung, after he had had x-ray pictures made in his own office and diagnosed them himself. An x-ray man was called in to see the films without knowing whose chest they represented and confirmed the diagnosis.

The American Legion was ever near to the heart of Dr. Dunn, who is said to have spent large sums annually in the work of that organization and disabled veterans.

Dr. Dunn was a member of the Phi Delta Theta Fraternity in college; later he became a Thirty-Second Degree Mason; while in religion he was a member of the Presbyterian Church.

At his own request his body was wrapped in an American flag and he received a military burial conducted by the American Legion.

Dr. Dunn was a man who by ability and training could have graced a university chair with distinction; and one regrets that students could not have come within the circle of his influence.

The South and the Nation have lost a brave, honest man, a profound student and a brilliant clinician; while the Southern Medical Association has suffered the loss of a Vice-President who was one of its outstanding members.

ALABAMA

Deaths

Dr. Lincoln Laconia Burwell, Selma, aged 61, died suddenly March 6 of cerebral hemorrhage.

Dr. Porter Mixon, Prichard, aged 35 died May 14 of erysipelas.

ARKANSAS

The Leo N. Levi Memorial Hospital, Hot Springs, has received a donation of $15,000 from Louis M. Steinberg. This with a sum already held by the Hospital will make possible the erection of a clinic building.

At the Golf Tournament held at El Dorado during the meeting of the Arkansas State Medical Society, the Dewel Gann, Jr. Cup for 1928 was won by Dr. F. Walter Carruthers, Little Rock.

Dr. A. C. Shipp, Little Rock, has been appointed as a member of the Board of Trustees of the Arkansas Tuberculosis Sanatorium, filling the vacancy caused by the death of Dr. George S. Brown, Conway.

Dr. Frank A. Norwood, Ashdown, recently accepted a position with the U. S. Marine Hospital No. 43, Ellis Island, N. Y.

Deaths

Dr. James H. Foster, Fort Smith, aged 69, died May 1 of pneumonia.

Dr. George Snider Brown, Conway, aged 75 died May 11 of pneumonia.

DISTRICT OF COLUMBIA

The American Ophthalmological Society met in Washington recently. The following officers were elected: Dr. Walter R. Parker, Detroit, Mich., President; Dr. Emory Hill, Richmond, Va., secretary. The next meeting will be held at Hot Springs, Va., in June, 1929.

Former representative John Dalzell left $10,000 to the Homeopathic Hospital, Washington, as a memorial to his wife.

Deaths

Dr. Albert Wilgus, Washington, aged 82, died April 27 at Brooklyn of myocarditis.

FLORIDA

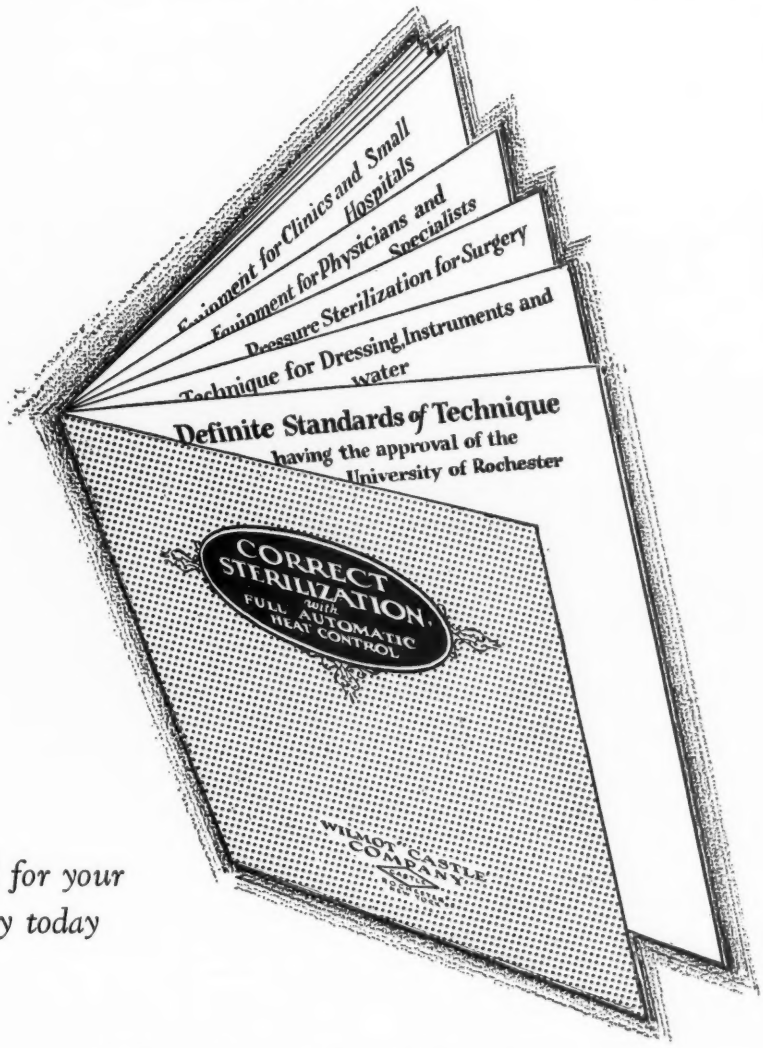
It has been announced that Governor Martin has taken steps to carry out the provisions of a 1927 legislative act for the establishment of a state tuberculosis sanatorium in Florida. The following have been named as the three members of the State Tuberculosis Board to conduct a survey of the state to determine the most suitable location for erection of the proposed sanatorium: D. B. McKay, Tampa, newspaper publisher and Mayor of that City; Lewis M. Lively, Tallahassee, and John L. Edwards, Ocala. The act appropriates $200,000 for establishment and maintenance of the sanatorium.

Deaths

Dr. Thomas Fraser Thomas, Gainesville, aged 69, died suddenly, April 29 of angina pectoris.

(Continued on page 34)

An Authoritative Publication



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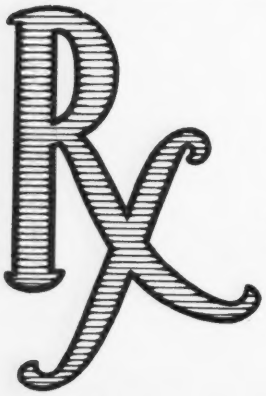

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(Continued from page 682)

Dr. Clarence H. Vaught, St. Petersburg, aged 62, died May 13 at the Mound Park Hospital of chronic nephritis.

Dr. William P. McKee, Eustis, aged 90, died April 21 of hemiplegia.

GEORGIA

At the recent annual meeting of the Georgia Tuberculosis Association held in Atlanta, Mr. Lee M. Happ, Macon, was elected President; Dr. I. A. White, Cartersville, and Mr. R. W. Hatcher, Milledgeville, Vice Presidents; Miss Virginia Gibbes, Marietta, Secretary; Mr. T. K. Glenn, Atlanta, Treasurer. The Secretary and Treasurer were reelected.

The Seventh District Medical Society met in Cedartown April 4. Dr. R. M. Harbin, Rome, was elected President; Dr. C. V. Wood, Cedartown, Vice-President. Dr. M. M. McCord, Rome was elected Secretary last April for a period of two years.

Decatur County Medical Society has elected Dr. E. C. Smith, Donalsonville, President; Dr. W. L. Wilkinson, Bainbridge, Vice-President; Dr. John I. Spooner, Donalsonville, Secretary-Treasurer.

Douglas County Medical Society has elected Dr. C. V. Vansant, Douglasville, President; Dr. J. T. Henley, Douglasville, Vice-President; Dr. D. Housworth, Douglasville, Secretary-Treasurer.

Habersham County Medical Society has elected Dr. J. H. McClure, Cornelia, President; Dr. W. H. Garrison, Clarkesville, Vice-President; Dr. R. B. Lamb, Demorest, Secretary-Treasurer.

Lamar County Medical Society has elected Dr. J. A. Corry, Barnesville, President; Dr. J. M. Rogers, Barnesville, Vice-President; Dr. John M. Anderson, Barnesville, Secretary-Treasurer.

Screven County Medical Society has elected Dr. L. F. Lanier, Sylvania, President; Dr. W. R. Lovett, Sylvania, Vice-President; Dr. W. W. Evans, Halcyondale, Secretary-Treasurer.

Truetlen County Medical Society has elected Dr. Geo. M. Barwick, Soperton, President; Dr. O. B. Moye, Soperton, Vice-President; Dr. L. I. Lanier, Soperton, Secretary-Treasurer.

Walton County Medical Society has elected Dr. H. L. Upshaw, Social Circle, President; Dr. J. B. H. Day, Social Circle, Vice-President; Dr. J. K. McClintic, Monroe, Secretary-Treasurer.

The staff of McCall Hospital, Rome, has elected the following officers: Dr. J. C. Watts, President; Dr. W. G. Barnett, Vice-President; Dr. R. O. Simmons, Secretary. The annex to the Hospital has been completed.

The Second District Medical Society will hold its next meeting at Camilla in November.

The Nolan Sanitarium and the Marietta Hospital, Marietta, have merged and will occupy the new hospital building which will soon be completed. Dr. W. Mayes Gober has been elected Chairman of the Board of Directors and will serve as Chief Surgeon. A bronze tablet perpetuating the memory of Dr. Warren E. Benson, President of the hospital association at the time of his death, will be placed at a conspicuous place at the front of the Marietta Hospital.

The Rockefeller Foundation has selected Macon as one of the twelve cities in which to train its students in carrying out health programs. Among the first to visit Macon will be Drs. Julio Freijanes and Rodrigo, Provisional Health Officers of the National Health Service of Spain.

Drs. W. H. Perkinson, Marietta, and M. M. McCord, Rome, have been appointed as members of the official staff of Governor L. G. Hardman, with the rank of Lieutenant-Colonel.

Dr. S. L. Cheshire, Thomasville, has been elected as a member of the County Board of Health.

Dr. E. C. Thrash, Atlanta, has been elected Chairman of the Reference Committee of the American Medical Association.

Dr. Wm. A. Mulherin, Augusta, has been elected to membership in the American Pediatric Society. It has been stated that he is the first Georgian to be elected to membership in this Society and that there are only eighty-five members in America.

Dr. H. P. McDonald has been elected President of the Fulton High School Alumni Association.

Dr. Matthew C. Baines, for the past seven years Commanding Officer of the U. S. Veterans' Bureau

(Continued on page 36)

X-rays Precede Appendectomies

The diagnosis which precedes an appendectomy includes the observations of clinical symptoms, a blood count, palpation and a well-made, properly interpreted x-ray examination. The x-ray examination becomes increasingly important as the symptoms and signs of palpation become less definite. An appendix may act as a focus of infection for a considerable time without giving rise to marked physical pain. Therefore even when not specifically indicated, gastro-intestinal x-rays often point out the difficulty most definitely.

The gastro-intestinal region may be the cause of many distressing symptoms. Palpation, percussion and auscultation, after all, lack much of the definiteness of the inherent accuracy of radiographic examination, which shows not only the *existence* of pathology but its *extent*. The locale is precisely fixed.

But with all its advantages, the x-ray examination may fall far short of its capabilities if the recording medium, the x-ray film, is not able to record the shadow details. Careful diagnosticians always look for the words "Eastman" and "Kodak" on the edges of the developed films. These assure them that Eastman Film was used. With proper technic and competent interpretation Eastman Film means radiography at its best. It is supplied in two types: *Super-Speed* and *Contrast*.

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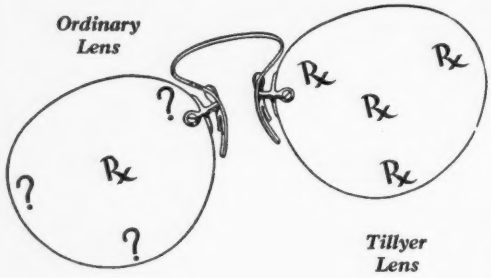

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(Continued from page 34)

Hospital at Augusta, has been transferred to a similar position at the Veterans Bureau Hospital in Bronx, New York.

Dr. Leon E. Brawner, Atlanta, and Miss Francis L. Winters were married at the home of the bride's parents at Alliston, Mass., June 20.

Dr. Roger W. Dickson and Miss Enid Boyce, Atlanta, were married at Emory University chapel June 15.

Deaths

Dr. Albert Butts Hinkle, Macon, aged 62, died February 14, at San Diego, Calif., of cerebral hemorrhage, chronic nephritis and myocarditis.

Dr. Jacob Lee Mathews, Hawkinsville, aged 54, died April 18.

Dr. Philip Hamilton Fitzgerald, Blakely, aged 47, died suddenly April 30.

Dr. Henry A. Turner, Rome, aged 77, died May 3 of senility.

Dr. George Washington Julian, Tifton, aged 70, died May 29 of cholecystitis.

Dr. N. T. Carswell, Macon, aged 67, died May 1 of angina pectoris.

KENTUCKY

The next annual meeting of the Kentucky State Medical Association will be held in Richmond, September 10-13.

Dr. Adolph O. Pfingst has resigned as Professor of Ophthalmology, University of Louisville, and as Director of the Eye Clinic at the Louisville City Hospital. Dr. Pfingst has been associated with the Medical Department in the capacity of teacher since 1896.

Deaths

Dr. Campbell H. Johnson, Paducah, aged 57, died suddenly May 4 of heart disease.

Dr. Charles A. Niles, Dawson Springs, aged 56, died May 16 in a hospital at Chicago of brain abscess following a frontal sinus infection.

Dr. Travis Alexander Bullington, Louisville, aged 67, died April 9 of chronic bronchiectasis.

Dr. Samuel Johnson Hall, Louisville, aged 62, died April 29 of intestinal obstruction.

Dr. Edward F. Weeks, LaGrange, aged 67, died May 22 as the result of a cerebral hemorrhage.

Dr. John Fleming Irvin, Tollesboro, aged 74, died May 2 of pneumonia.

Dr. Lonzo Harrison Redman, Chevrolet, aged 53, died May 10 of cerebral hemorrhage.

Dr. Charles Duerson, Mount Sterling, aged 89, died May 10 of chronic myocarditis and renal insufficiency.

LOUISIANA

The New Orleans Gastro-Enterological Society was recently organized. The following officers were elected: Dr. Sidney K. Simon, President; Dr. J. A. Storck, Vice-President; Dr. A. L. Levin, Secretary-Treasurer.

At a recent meeting of the Board of Administrators of the Tulane University of Louisiana, New Orleans, the name of Dr. Henry Daspit was approved for appointment as Dean of the Graduate School of Medicine, effective September 1.

A branch laboratory of the State Board of Health has been opened in Alexandria as a part of the Rapides Health Unit. The State Board of Health furnishes the equipment and material for its use; the Parish pays the technician's salary and furnishes the quarters, and the City furnishes the utilities, gas, water and electricity.

Dr. Hiram W. Kostmayer, New Orleans, has been appointed Professor of Gynecology and head of this department in the Graduate School of Medicine of Tulane University.

Dr. Ansel M. Caine, New Orleans, has been elected President of the Associated Anesthetists of the United States and Canada.

Dr. Eugene Davis, Medical Officer-in-charge of the U. S. Veterans' Hospital, New Orleans, was presented with a ring by the patients as a token of their esteem previous to his departure to Memphis, Tenn., where he has been assigned to duty at U. S. Veterans' Hospital Number 88.

MARYLAND

The Colonial Hospital, Baltimore, known during the World War as the Morrow Hospital, when in use

(Continued on page 38)

Coax the convalescent's appetite with tempting, health-building dishes made from KNOX SPARKLING GELATINE

BECAUSE of its easy digestibility and protein value, Knox Sparkling Gelatine has been given a high dietetic position, particularly in the regimens of convalescents, the gastro-enterically delicate, and of anorexic patients. As a vehicle for fresh fruits and vegetables, eggs, milk, and in jellied meat preparations, Knox Sparkling Gelatine permits a number of variations in the prescribed diet—dishes which are both appetizing in appearance and satisfying as to bulk.

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Manhattan Eye Salve Company

Louisville, Kentucky

(Continued from page 36)

by the U. S. Public Health Service, was closed June 1. The owner has decided to limit his work to private practice and to sell the three hospital buildings and their equipment.

The addition to Eudowood Sanatorium, made possible by a gift from Mrs. Mary Frick Jacobs, was dedicated June 2. The unit will house 40 tuberculous children and is a testimonial from Mrs. Jacobs to her husband, Dr. Henry B. Jacobs, President of the Board of Directors of the institution.

By the will of Mrs. Rebecca Lanier King, Baltimore, the Johns Hopkins University is to receive $50,000, the Johns Hopkins Hospital $105,000, the Presbyterian Eye, Ear and Throat Charity Hospital $5,000, and St. Joseph's Hospital $2,500. These hospitals are in Baltimore.

Dr. Howard A. Kelly's mycologic library, said to be the most complete and valuable of its kind privately owned in the United States, has been offered to the regents of the University of Michigan and accepted. The collection will be called the Louis C. C. Krieger Mycological Library and Collection Herbarium at Dr. Kelly's request to memorialize the Baltimore artist and mycologist who gathered the library during the last ten years for Dr. Kelly. The collection will be housed in a new $800,000 building with three other collections pertaining to the natural sciences.

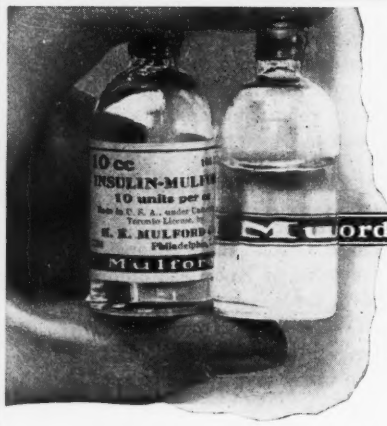
The Henry Phipps Psychiatric Clinic celebrated its 15th anniversary April 30.

Dr. Paul Preble, Baltimore, who has been loaned to the State Department of Health by the U. S. Public Health Service and has been acting as temporary head of the Bureau of Vital Statistics, has been relieved of that work and will make a study of the bureaus of the Department and recommendations for improvement to the State Board of Health. Dr. John Collinson, Baltimore, has been made Acting Head of the Bureau of Vital Statistics.

Dr. Thomas R. Brown, Baltimore, has been reelected President of the Johns Hopkins Medical Alumni Association.

Dr. William S. Thayer, Baltimore, President

(Continued on page 40)



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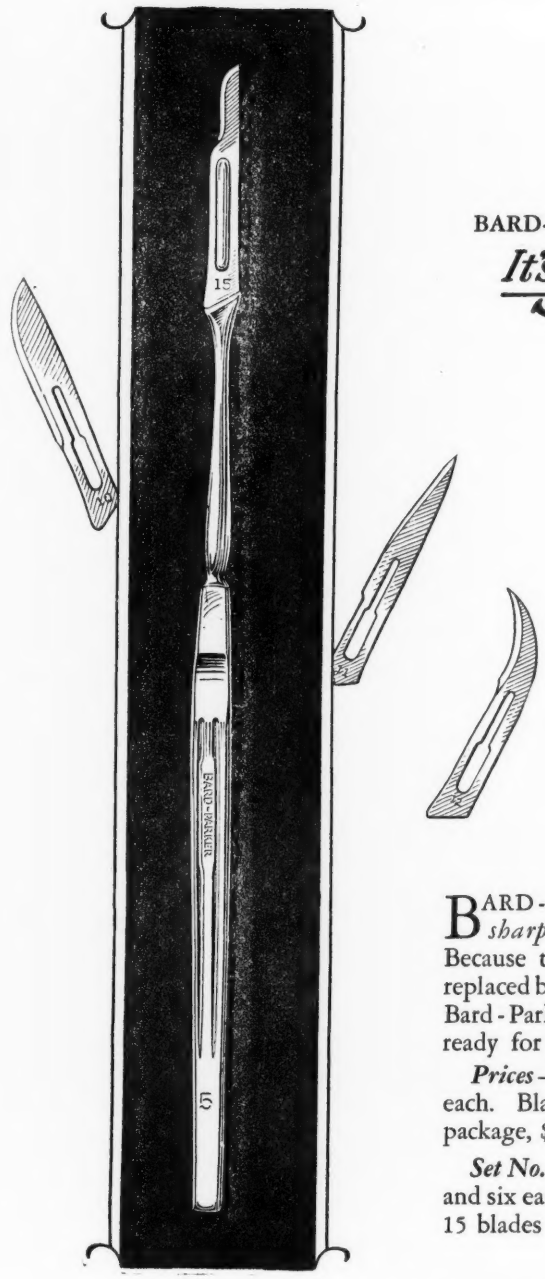

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(Continued from page 38)

of the American Medical Association, was a delegate to the Harvey Tercentenary celebration in London in May.

Dr. John Warton Harris, Associate Professor of Obstetrics, Johns Hopkins University School of Medicine, Baltimore, has resigned to become Professor of Obstetrics and Gynecology at the University of Wisconsin Medical School, Madison. Dr. Sydney W. Britton, Associate in Physiology at Johns Hopkins, has resigned to become Professor of Physiology at the University of Virginia Department of Medicine, Charlottesville, and Dr. Phyllis Greenacre has resigned as Associate in Psychiatry.

Dr. Perry F. Prather, Hagerstown, has been reappointed Health Officer of Washington County.

Deaths

Dr. Arthur Pendleton Herring, Baltimore, aged 53, died May 29 of carcinoma.

Dr. David L. Gifford, North East, aged 77, died May 21 at a hospital in Port Deposit, of carcinoma of the cardiac end of the stomach and diabetes mellitus.

Dr. William Edward Wiegand, Baltimore, aged 75, died suddenly May 13 of angina pectoris.

Dr. Charles Percy Kemp, Baltimore, aged 59, died April 8 of facial erysipelas and lobar pneumonia.

MISSISSIPPI

The regular quarterly meeting of the Northeast Mississippi Thirteen Counties Medical Society was held at Houston, June 19.

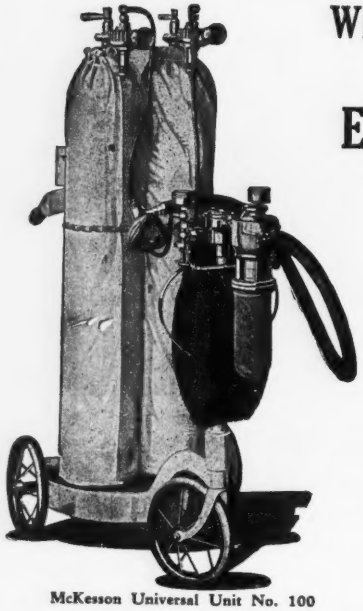
Deaths

Dr. W. W. Weathersby, Fair River, aged 60, was found dead in his pecan grove near his residence May 19.

Dr. William M. Murry, Ripley, aged 76, died April 16 of influenza.

Dr. Burris Earle Burchfield, Ruleville, aged 39, died April 19 at a hospital in Greenville.

(Continued on page 42)



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GRASS HAYFEVER begins about the time *tree hayfever* ends, *viz.* May 15th, and need not be confused with the earlier appearing and sometimes overlapping tree hayfever.

WEED HAYFEVER—August to frost—is unrelated to the previously occurring *grass hayfever* and is occasioned, according to the locality, by such late pollinating plants as the Ragweeds—Russian Thistle—Western Water Hemp—Carelessweed—or Sage Brush.

LIST of pollens for any section—any season—with commentary circular discussing the treatment of hayfever by *preseasonal* or *coseasonal* method, with respective schedules of dosage—sent on request.

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(Continued from page 40)

MISSOURI

At the recent annual meeting of the Missouri State Medical Association held at Jefferson City, Dr. Frank I. Ridge, Kansas City, was installed as President; Dr. Tolman W. Cotton, Van Buren, elected President-elect; Dr. George W. Hawkins, Salisbury, Treasurer, re-elected; Dr. Edward J. Goodwin, St. Louis, Secretary, reelected. The next meeting will be held at Springfield.

The Missouri Public Health Association held its fourth annual meeting at Jefferson City, May 5. Dr. W. W. Johnston, Jr., Farmington, was elected President; Dr. John W. Williams, Jr., Springfield, Vice-President.

The fall clinical conference of the Kansas City Southwest Clinical Society will be held at Kansas City, October 9-11.

Christian County Medical Society has elected Dr. H. J. Wise, Sparta, President; Dr. J. H. Wade, Ozark, Vice-President; Dr. F. H. Brown, Billings, Secretary-Treasurer.

Howard County Medical Society has elected Dr. W. R. Hawkins, Glasgow, President; Dr. W. J. Shaw, Fayette, Vice-President; Dr. W. A. Bloom, Fayette, Secretary-Treasurer.

Health Commissioner Starkloff, St. Louis, announces that plans are being made to equip the ten health centers in St. Louis for diagnostic cancer clinics. They will be under the direction of the board of visiting nurses, and physicians now in the diagnostic division of the Health Department will assist. The clinic will be for diagnostic purposes only and will become a permanent part of the health center work.

A $50,000 addition to the Odd Fellows' State Hospital, Liberty, was dedicated by officers of the Grand Lodge in the home at Liberty in the annual homecoming May 13.

An appropriation of $100,000 has been made by the St. Louis County Court for a 28½ acre site in Clayton for the $1,000,000 St. Louis County General Hospital for which a bond issue was recently voted.

About $12,000 was realized from the annual country carnival given in the interest of the proposed St. Louis Charity Hospital at the country home of Mr. and Mrs. Samuel W. Fordyce, Kinloch, St. Louis County.

The proposition of the St. Louis Medical Society and the Trudeau Club for a $2,000,000 bond issue, to cope with an acute shortage of city facilities for the care of the tuberculous, is meeting with an intelligent appreciation by the city officials.

The Administrative Board of St. Louis University Medical School was recently announced as follows: Rev. Alphonse M. Chwitalla, S. J., Dean; Dr. Don R. Joseph, Associate Dean; Dr. John Auer, Professor of Pharmacology; Dr. William T. Coughlin, Professor of Surgery; Dr. Ralph A. Kinsella, Professor of Internal Medicine; Dr. James P. Harper, Dean of the Dental School.

Edmund V. Cowdry, Ph.D., of the Rockefeller Institute for Medical Research, New York, was recently appointed Professor of Cytology in Washington University Medical School, St. Louis.

Dr. Joseph Grindon, head of the Dermatology Department of St. Louis University, was elected President of the American Dermatological Association, Washington, D. C., recently.

Dr. David P. Dyer, Sedalia, has been appointed Director of the Free Emergency Hospital at the Missouri State Fair August 18-25. This is Dr. Dyer's seventeenth year to be in charge of the State Fair's Hospital.

Dr. Harvey J. Howard has been appointed full-time Professor of Ophthalmology in the Washington Uni-

(Continued on page 44)

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POWDERED PROTEIN MILK

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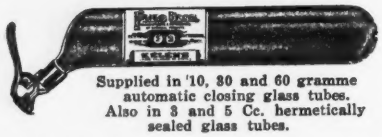

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(Continued from page 42)

versity Medical School, St. Louis, and Jacques J. Bronfenbrenner, Dr. P. H., has been appointed Professor and Head of the Department of Bacteriology and Immunology.

Dr. Lee Wallace Dean, formerly Dean and Professor of Otolaryngology and Oral Surgery, of the State University of Iowa College of Medicine, Iowa City, has been appointed full-time Professor of Otolaryngology at Washington University Medical School, St. Louis.

Dr. W. O. Coleman, Joplin, and Mrs. Ida Harris, Decatur, Ill., were married recently.

Deaths

Dr. Meyer J. Lippe, St. Louis, aged 51, died March 22.

Dr. William H. Carruthers, St. Louis, died April 22.

Dr. Sophia L. Kern, St. Louis, aged 72, died April 10 at the home of her daughter in Kansas City.

Dr. Lawrence Patrick Fox, Weston, aged 36, died May 7 in a hospital at St. Joseph of pneumonia.

Dr. Franklin G. Weary, St. Joseph, aged 66, died suddenly May 2 of cerebral hemorrhage.

Dr. William R. Goodykoontz, Desloge, aged 80, died suddenly May 7 of heart disease.

Dr. John M. Robinson, Liberty, aged 81, died April 27 of prostatitis.

Dr. Quintin Moore Brown, Nevada, aged 40, died April 28 of transverse myelitis following influenza.

NORTH CAROLINA

A new medical building will be erected for Wake Forest College, Wake Forest, in memory of Dr. William A. Johnson, formerly Professor of Anatomy at the College.

The new Babies Hospital, Wrightsville Sound, has been opened, according to the announcement of Dr. J. Buren Sidbury.

Edward M. Harris, Philadelphia, Pa., a native of North Carolina, plans to erect a 100 bed modern hospital in Raleigh at a cost of more than $300,000, directed by a Board of Trustees, this in memory of his father who for several years was Editor of the Orange County Observer, published at Hillsboro. It will be a general hospital, supported by endowment, and will admit charity and pay patients.

Dr. E. S. King was recently made Professor of Bacteriology and Physiological Chemistry, Wake Forest College.

Dr. Hamilton W. McKay, Charlotte, has been chosen a Trustee of Davidson College, and President of the College's Alumni Association.

Dr. William Preston Holt, Jr., Erwin, and Miss Flora Gray Jerome, Goldsboro, were married May 31.

Dr. Edward Landling King, Wake Forest, and Miss Helen Valentine Dunn, Wake Forest, were married June 2.

Dr. R. K. Farrington and Miss Mary Louise Bailey, both of Thomasville, were married May 26.

Dr. George Grady Dixon, Ayden, and Miss Juliana Elliott, Hertford, were married at Fayetteville March 24.

Dr. Charles Glenn Poole, Winston-Salem, and Miss Audrey Allen Brenegar, Mocksville, were married April 18.

Dr. John William Roy Norton, Laurinburg, and Miss Juanita Ferguson, Jackson, Miss., were married April 8.

Deaths

Dr. Eugenius A. Hall, Statesville, aged 89, died June 2.

Dr. Wallace E. Coltrane, Dunn, died June 2 in a hospital at Smithfield.

Dr. J. B. Smith, Pilot Mountain, aged 67, died at a hospital at Winston-Salem May 31, following an illness of several weeks with Bright's disease.

Dr. Benjamin F. Halsey, Roper, died June 15.

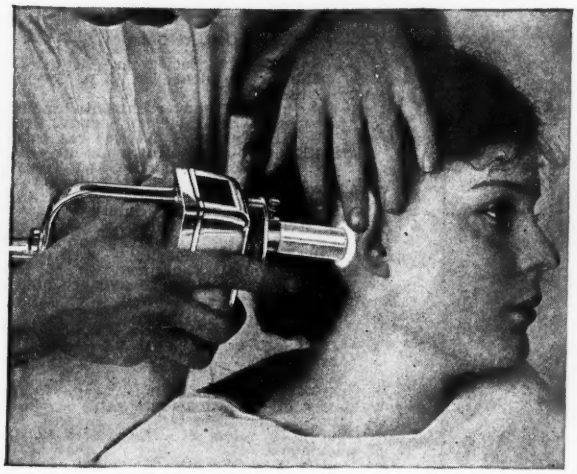
Dr. Bartlett J. Witherspoon, Charlotte, died suddenly June 9.

Dr. William Roy Butler, Boone, aged 71, died April 30 of carcinoma of the stomach.

Dr. James P. Battle, Nashville, aged 60, died April 16 as the result of paralysis.

Dr. Thomas Irvin Burbage, Como, aged 65, died April 15 of cerebral hemorrhage.

(Continued on page 46)



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"Ultra-violet light from the air-cooled mercury vapor quartz lamp is a specific in tetany, rickets, and spasmophilia, and is very valuable as a general tonic in depressed bodily states such as anorexia and some cases of anemia, and in poor nutrition . . ." The Atlantic Medical Journal, Vol. XXXI, No. 8, May, 1928, pages 537-542.

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Each burner must pass a rigid test in the Hanovia laboratories, to insure the highest intensity and the longest service.

The economy of the *Hanovia* Burner is apparent in its low consumption of current.

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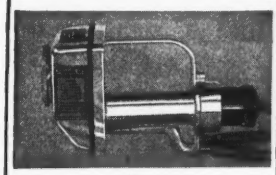

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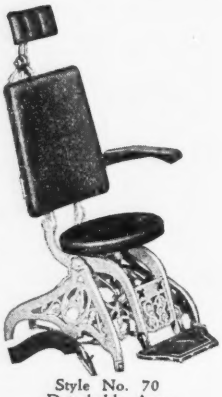

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(Continued from page 44)

OKLAHOMA

Noble County Medical Society has elected Dr. F. F. Renfrow, Billings, President; Dr. B. A. Owens, Perry, Secretary-Treasurer.

At the one hundred and seventeenth annual meeting of the Rhode Island Medical Society, Providence, June 7, the Fiske prize of $250.00 was awarded to Dr. Charles R. Rayburn, Norman, for his essay "Etiology, Symptomatology, Diagnosis and Treatment of Epidemic Encephalitis," submitted during the last year. The Fiske contest is conducted annually and is open to any physician in the country.

Dr. J. A. Muller, Snyder, has been appointed Physician in the U. S. Veteran's Bureau and has been assigned to the Muskogee Hospital.

Dr. Carl F. Jordan, Superintendent of Health of Muskogee County, has resigned and accepted a position as Professor of Hygiene in the University of Iowa.

Dr. V. C. Tisdal and Miss Ina McDaniel, Elk City, were married May 5.

Deaths

Dr. Charles C. Sims, Seminole, aged 52, died suddenly May 9 in his office following a stroke of apoplexy.

Dr. Duncan H. Selph, Stillwater, aged 69, died May 19 of pneumonia.

Dr. Hardin Lee Washer, Hollister, aged 64, died April 26.

Dr. Julius Eugene Farber, Cordell, aged 64, died suddenly in May at El Reno, of heart disease.

Dr. Robert S. Bonner, Fort Towson, aged 70, died May 12 of heart disease.

Dr. James Johnson Williams, Weatherford, aged 60, died in May at a hospital in Clinton.

SOUTH CAROLINA

South Carolina's laboratory, in which tests will be made to determine the mineral and medicinal content of vegetables and farm produce grown in the State, will be located in Charleston at the Medical College of South Carolina, it was announced by Dr. Robert Wilson, Dean of the Medical College and a member of the State Food Analysis Commission. The selection of a location for the laboratory was left in the hands of Dr. Roe E. Remington, Minneapolis chemist, employed by the Commission. Dr. Remington has been elected a member of the Faculty of the Medical College with the title of Professor of Nutrition and Director of the Food Research Laboratory. The tests were authorized by act of the General Assembly creating the food analysis commission, of which Dr. William Weston, Columbia, is Chairman, and appropriating $15,000 to finance the analysis of South Carolina grown foods and feedstuffs with the idea of determining their content in iodin, calcium and other minerals to the presence of which is attributed the practical absence of goiter in the State.

Deaths

Dr. James Davison McDowell, York, aged 54, died June 3.

TENNESSEE

Dr. G. Canby Robinson, Nashville, Dean of Vanderbilt School of Medicine, has been appointed Dean and Director of the New Medical Center to be constructed by Cornell University Medical College and the New York Hospital, New York. Dr. Waller S. Leathers, Dean of the School of Public Health, Vanderbilt, has been selected as Dr. Robinson's successor as Dean of Vanderbilt School of Medicine.

The annual meeting of the Tennessee Association of Railroad Surgeons met in Memphis April 10. The following officers were elected: Dr. Duncan Eve, Jr., Nashville, Chairman; Dr. Britt Burns, Memphis, Vice-Chairman; Dr. A. F. Richards, Sparta, Secretary.

The East Tennessee Medical Society has elected Dr. James S. Lyons, Rogersville, President; Dr. Jesse C. Hill, Knoxville, Secretary.

Middle Tennessee Medical Asociation met at Franklin May 11. The following officers were elected: Dr.

(Continued on page 48)

What is Psylla?

IN the *correction* of constipation two factors are essential to permanent results, namely bulk and lubrication.

A rare and little known agent in constipation is

PSYLLA

the seed of a plant (plantago psyllium) which grows wild in Asia Minor. This seed, especially selected and sterilized by us in Battle Creek, provides both bulk and lubrication.

The action of Psylla is purely mechanical. It is an accessory food. On contact with moisture it swells, giving a bland, non-irritating bulk and lubrication in the intestine.

We shall be glad to send you a can of Psylla for test without charge.

Combat Intestinal Toxemia

And putrefaction—a frequent cause of serious disease and a grave handicap in all diseases—by the use of the colon food

LACTO-DEXTRIN

This food product, with a therapeutic purpose, promotes the growth of the benign bacteria in the intestine. It is pleasant to take and definite in action.

Fully described in the scientific presentation "The Intestinal Flora". Write for a copy today.

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NEW ORLEANS, LA.

(Continued from page 46)

Sam P. Bailey, Nashville, President; Dr. Dan German, Jr., Franklin, Vice-President; Dr. Murray B. Davis, Nashville, Secretary. The November meeting will be held at Columbia.

The new contagious hospital, a unit of the Knoxville General Hospital, Knoxville, has been completed and is ready to receive patients.

Remodeling of the Crook Sanitarium, Jackson, has been completed and the institution is now equipped to handle ninety-five patients.

At the commencement of Meharry Medical College, Nashville, May 24, a gift of $18,500 from the senior class to the endowment fund was announced. Starting three years after graduation, thirty-seven members of this class will pay $25.00 annually for twenty years.

The annual banquet of the Vanderbilt chapter of Alpha Omega Alpha, Nashville, was held May 15. The following alumni were initiated: Dr. Eugene L. Bishop, State Health Officer; Dr. Ira R. Sisk, Madison, Wisc.; Dr. Beverly T. Nolan, Franklin; Dr. Henry Clay Long, Knoxville; Capt. Joe H. St. John, U. S. Army Medical Corps.

A Children's Clinic was held at the Rutherford Hospital, Murfreesboro, May 2-3. The visiting clinicians were: Dr. Seale Harris, Birmingham, Ala.; Dr. Horton R. Casparis, Nashville; Dr. Lesesne Smith, Spartanburg, S. C.

Dr. J. Walter McMahan has resigned as Medical Director of the Aluminum Company of America, Alcoa, on account of ill health.

Dr. G. D. Lequire has been elected Chief of Staff for the Mountain Sanitarium, the new tuberculosis hospital for children at Maryville.

Dr. Lorenz A. Miesch, Bolivar, recently accepted a position at the Feebleminded Institute at St. Louis, Mo.

Deaths

Dr. Vernon C. Lane, Knoxville, aged 39, died May 26.

Dr. W. E. Harwell, Frankewing, aged 52, died May 19 of apoplexy.

Dr. Walter W. Graham, College Grove, aged 59, died May 23 of pneumonia following influenza.

Dr. James Dick Bridger, Memphis, aged 56, died May 9 at Bolivar.

TEXAS

At the annual meeting of the State Medical Association of Texas held at Galveston May 8-10, the following officers were elected: Dr. Joseph E. Dildy, Brownwood, President-Elect; Dr. D. H. Hudgins, Forney, Dr. Stephen D. Naylor, Stephenville, and Dr. Job L. Hammond, Paris, Vice-Presidents; Dr. Klever H. Beall, Fort Worth, Treasurer; Dr. Holman Taylor, Fort Worth, reelected Secretary. The next meeting will be held at Brownsville.

The meeting of the Texas Radiological Society was held at Galveston May 7. Dr. Davis Spangler, Dallas, was elected President; Dr. J. B. Johnson, Galveston, President-Elect; Dr. C. F. Lehmann, San Antonio, and Dr. W. G. McDeed, Houston, Vice-Presidents; Dr. C. P. Harris, Houston, Secretary-Treasurer.

(Continued on page 50)

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"RIEDEL"
The Distinctive Properties of Gonosan
Inhibits gonococcal development and minimizes its virulence.
Aids in reducing the purulent secretion.
Encourages normal renal activity.
Relieves the pain and strangury and allays the irritation and inflammation.
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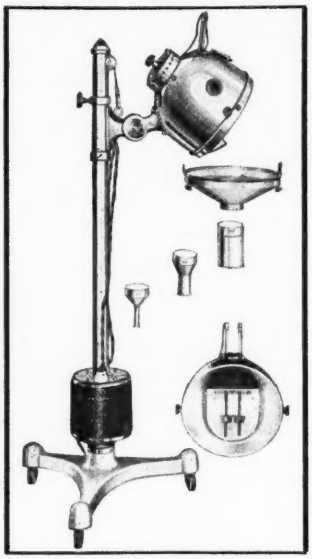

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(Continued from page 48)

The meeting of the Texas Railway Surgeons Association was held at Galveston May 7. Dr. S. P. Cunningham, San Antonio, was elected President; Dr. D. M. Higgins, Gainesville, and Dr. J. J. Robertson, Kingsville, Vice-Presidents; Dr. Ross Trigg, Fort Worth, Secretary-Treasurer.

The meeting of the Texas Pediatric Society was held May 9 at Galveston. The following officers were elected: Dr. Edwin Schwarz, Fort Worth, President; Dr. George Cornick, San Antonio, Vice-President; Dr. Ramsey Moore, Dallas, Secretary-Treasurer.

The Texas Neurological Society was organized May 8 at Galveston. The following officers were elected: Dr. John S. Turner, Dallas, President; Dr. James Greenwood, Houston, Vice-President; Dr. W. L. Allison, Fort Worth, Secretary-Treasurer.

The Texas Dermatological Association was organized May 8 at Galveston, Dr. S. J. Wilson, Fort Worth, was elected President; Dr. Porter Brown, Fort Worth, Secretary. Meetings will be held semi-annually, one at the time of each annual session of the State Medical Association, and the other at some date to be decided upon during the year.

Dr. Theodore Johnson, Glen Rose, is planning to erect a sanitarium of not less than 25 rooms for patients.

Dr. C. B. Williams, Mineral Wells, has recently let the contract for the construction of a clinic and hospital building at an approximate cost of $35,000.

The John Sealy Hospital, Galveston, plans the construction of a new out-patient clinic building estimated at a cost of $250,000. This new construction is a part of a $1,000,000 building program for the expansion of the facilities of the John Sealy Hospital, covering a two year period.

Dr. J. W. Gooch, Shamrock, is building a hospital at an approximate cost of $75,000.

Kings Daughters Hospital, Temple, has been enlarged and the Clinic reorganized. The new buildings were opened for public inspection May 12-13. Music was furnished by the Temple Baby Band composed of children under 6 years of age. The number of rooms for patients has been increased to 116.

Dr. W. P. White, Henderson, has been appointed Assistant Councilor for the Eleventh District.

Dr. Edward Randall, Sr., has resigned as Professor of Materia Medica and Therapeutics in the Medical Department of the University of Texas, Galveston. He had had thirty-eight years of active service with the institution. In appreciation of his untiring work with the Medical College, the Board elected him President Emeritus of the institution and gave him a seat on the Faculty and on the Executive Committee of the College, as long as he lives.

Dr. and Mrs. S. E. Thompson, Kerrville, left June 2 for New York. They sailed on June 16 for Europe where Dr. Thompson is to join the Foreign Assemblies Clinic this year.

(Continued on page 52)

CLASSIFIED ADVERTISEMENTS

ATTENTION SURGEONS AND DOCTORS wanting good location and sanitarium. For sale at once well built three-story brick forty-room furnished commercial hotel for less than half value. Time ripe for it to be turned quickly into a hotel sanitarium. Not even a city hospital here and no sanitarium in hundred miles. Population about 8000, backed by a thickly populated country. Large cotton mills, large silk mills and large granite mines. Many expert workers and every one else including doctors begging for a sanitarium to be opened here. Elberton is in the heart of the Piedmont Section, Northeast Georgia. Finest all the year round climate in the South. Three railroads, junction of several through highways. Too many hotels here and no hospital. Write or see D. Sheppard, Agent, Hotel Gholston, Elberton, Ga.

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LABORATORY AND X-RAY TECHNICIAN, two years' experience, desires position with clinical or hospital laboratory. Special training acquired in a hospital and medical school in x-ray technique, therapy and developing, also in hematology, serology, bacteriology and basal metabolism. Am college graduate. A1 references. A. B., care Southern Medical Journal.

MEDICAL HYDROLOGY—A series of 12 brochures on Mineral Water therapy in general; and the Bedford Mineral waters in particular, is being issued monthly, aggregating about 200 pages, for free distribution to the profession. We will be pleased to send the entire series, with a binder for permanent preservation, to physicians so requesting. William E. Fitch, M. D., Medical Director, Bedford Springs Hotel and Baths, Bedford, Penn.

GUINEA PIGS FOR SALE—For laboratories and pets. C. Kopp, Tracy City, Tenn.

DRUG AND ALCOHOLIC PATIENTS are humanely and successfully treated in Glenwood Park Sanitarium, Greensboro, N. C.; reprints of articles mailed upon request. Address W. C. Ashworth, M.D., Owner, Greensboro, N. C.

TO THE MEDICAL PROFESSION: We are filling a long-felt want by conducting a training class teaching blood counts, urinalyses, sputum and feces examinations; also basal metabolism readings. We will gladly furnish you with competent office assistants trained in such laboratory procedures as you may require. Our classes are made up of stenographers, bookkeepers, registered nurses and clerks. Send us your office assistant for training. Write or telephone for full information. Dial 3-3598. John V. Mix, Director, Alabama Pathological Laboratory, 1005 Martin Building, Birmingham, Ala.

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CENTRIFUGES

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(Continued from page 50)

Deaths

Dr. J. Frank Rayburn, Bonham, died May 19.

Dr. Clarence K. Haggard, Moody, died February 29 of cerebral anemia.

Dr. Keith Wilson Cameron, San Antonio, aged 36, died March 11 of bronchopneumonia.

Dr. Michael P. Fenelon, Jourdanton, aged 59, died suddenly March 1 of heart disease.

Dr. Camden P. Pence, Dallas, aged 48, died May 16 of carcinoma of the breast.

Dr. Philip Hill, San Antonio, aged 46, died suddenly May 8.

Dr. Sidney J. Smith, Houston, aged 59, died April 7.

VIRGINIA

The Southside Virginia Medical Association met in Richmond June 19.

The Association of Surgeons of the Southern Railway met at Virginia Beach in May. Dr. Walter S. Nash, Knoxville, Tenn., was elected President; Dr. George H. Searcy, Tuscaloosa, Ala., Dr. Arthur P. McElroy, Union, S. C., Dr. William H. Frampton, Charleston, S. C., and Dr. Samuel H. Hairston, Meridian, Miss., Vice-Presidents; Dr. William W. Harper, Selma, Ala., Recording Secretary; Miss Edith A. Foltz, Washington, D. C., Executive Secretary-Treasurer.

Fifty-four counties and nine cities in Virginia have have conducted an Early Diagnosis Campaign during the spring months. The work has been carried on under the auspices of the Virginia Tuberculosis Association.

The Johnston-Willis Interne Scientific Club was organized May 30 at Richmond. Dr. J. Morrison Hutcheson, Richmond, was elected President; Dr. F. S. Johns, Richmond, Secretary-Treasurer.

The Medical College of Virginia held its ninetieth commencement exercises for the schools of medicine, dentistry, pharmacy and nursing in Richmond, May 26-29.

(Continued on page 54)

Laboratories of

Drs. Bunce, Landham and Klugh

ATLANTA, GEORGIA

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(Continued from page 52)

Dr. W. A. Brumfield has resigned as Health Officer at the Virginia Polytechnic Institute, Blacksburg. He has become director of the nine-county Health Unit, composed of Prince Edward, Charlotte, Lunenburg, Amelia, Nottoway, Powhatan, Cumberland, Buckingham and Appomattox Counties.

Commander Micajah Boland, Medical Corps, U. S. Navy, has been detached from duty at Naval Operating Base, Hampton Roads, and assigned to duty on the U. S. S. Florida.

Dr. Goldsborough F. McGinnes has been appointed Director of Laboratories of the State Department of Health to succeed Mr. Aubrey H. Straus, who has resigned.

Dr. Charles R. Robins has been elected President of the Richmond Rotary Club.

Dr. Roshier W. Miller, Richmond, has been elected Chairman of the Richmond City School Board; Dr. Clifton M. Miller has been elected Vice-Chairman.

Dr. Raymond De Van Kimbrough, University, and Miss Janet Haldane Coleman, Williamsburg, were married May 5.

Deaths

Dr. Edward E. Field, Norfolk, aged 73, died May 29 after a long illness.

Dr. Frank Mason Dillard, Alexandria, aged 38, died in a hospital at Washington, D. C., May 18 after an illness of several months.

Dr. Paulus A. Irving, Farmville, aged 71, died June 11.

WEST VIRGINIA

Deaths

Dr. Gustavus Beall Hedges, Martinsburg, aged 71, died suddenly April 28, at the home of a relative in Baltimore, Md., of myocarditis.

Dr. Richard T. Stone, West Columbia, aged 59, died in May of paralysis.

Dr. Gilman R. Davis, Charleston, aged 74, died June 11 from injuries received in an automobile accident.

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